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MANGANESE PNEUMONITIS

BY

T. A. LLOYD DAVIES

From The Industrial Health Unit, Boots Pure Drug Co., Ltd., Nottingham

I. Clinical and Environmental Studies

Manganese dioxide (MnO_2) or pyrolusite is mined in Norway, Germany, India, Russia, Brazil and the Gold Coast. Manganese dioxide is employed in the manufacture of manganese steel, in the dry battery industry, for the colouring and bleaching of glass, and in the chemical industry. Basic slag contains about 10 per cent. of manganese.

Literature

Though Brezina (1921) reported that 5 out of 10 men working in a pyrolusite mill had died of pneumonia after working 2, 6, 3, 1 and 2 months, Baader (1932) was the first to ascribe to manganese the high incidence of pneumonia amongst workers making dry battery cells. Elstad (1939) observed a great increase in pneumonia among the inhabitants of Sauda, a small town in Norway, after the opening of works where manganese alloys are smelted. The mortality for pneumonia in Sauda was ten times greater than for the rest of Norway, and in Elstad's opinion was due to the pall of smoke, containing manganese dust, overhanging the town.

Buttner (1939) observed that men working in a Rhenish pyrolusite mine suffered an average pneumonia rate, for the years 1934–37 inclusive, of 17 (range 8–25) per thousand (with an average death rate of 6·3 per thousand). As a control group he examined the experiences of the Leipzig Local Sick Fund whose male members had a pneumonia rate of 0·54 per thousand. In Buttner's opinion the death rate was due to a mild silicosis which by fibrosis paved the way for the pneumonic process. The Giessen pyrolusite ore contains 17 per cent. manganese, 20 per cent. iron and 20 per cent. silicic acid, which probably accounts for the silicotic process observed by Buttner.

The Present Inquiry

Manganese acts in two ways: (1) manganese poisoning, characterized by changes in the central nervous system, and (2) by causing pulmonary symptoms commonly termed manganese pneumonia.

The present inquiry was the result of the observation that men employed in the manufacture of potassium permanganate, exposed to the inhalation of dust of manganese dioxide and higher oxides of manganese, suffered a high incidence of pulmonary symptoms, in particular illness diagnosed as pneumonia. Clinical and environmental studies were completed by the exposure of animals to the inhalation of manganese dioxide dust.

Description of Manufacturing Process

In the manufacture of potassium permanganate manganese dioxide is ground in a ball mill, 'reacted' with melted caustic potash (KOH) in the presence of lime (at a temperature of 400° C.) (fig. 1), roasted in rotary kilns (figs. 2 and 3) and subsequently in solution submitted to electrolysis (fig. 4) so that manganite, manganate and permanganate of potassium are formed in succession. Recovered manganese dioxide is dried in a roaster (fig. 5).

Of 34 batches of pyrolusite examined on delivery, 30 contained less than 2 per cent. of silica, and the remaining 4 batches less than 2·7 per cent. (with an average manganese dioxide content of 87–88 per cent.). Minor constituents are phosphorus pentoxide (0·2 per cent.), lead (0·002 per cent.), iron (3·5 per cent.), alumina (1·0 per cent.) and chromium (0·001 per cent.).

The work is heavy but the plant is not unduly hot (except during blackout) though the clothing needed to protect against caustic splashes adds to the heat and sweating (fig. 6). The cloakrooms are good and well supplied with hot water and fitted with shower baths, and from some parts of the plant can only be reached by going outside but no worker need walk more than 10 yards out of doors to reach the cloakroom. The majority need not leave the building. When 'reactions' are being done, a thick cloud of steam and dust, mostly manganese dioxide and lime, is given off, and at all times the plant is very dusty. Potassium manganate and permanganate spray is liberated in the electrolytic process (fig. 4). Except in special places no forced ventilation was provided though the plant was freely open to the outside air. Ventilation varies considerably with the direction of the wind, and by general consent is considered to be good when the wind is blowing from the west. This is partly because of the layout of the plant and partly because the most dusty jobs are on the east side.

Incidence of Illness diagnosed as Pneumonia

The incidence of illness diagnosed as pneumonia in a group of 40–124 men, exposed to the inhalation of manganese dust, varied from 15 to 63 (average 26) per thousand over the years 1938–45 inclusive, compared with an average rate of 0·73 per thousand for the years 1938–44 amongst the male members of Boots Health Insurance Society (Table 1).

In all cases the diagnosis of pneumonia in a manganese worker was made by the patient's medical attendant and confirmed by communication with the doctor. The first N.H.I. certificate of pneumonia received by Boots Health Insurance Society whose male members are, with few exceptions, equally divided between chemical and pharmaceutical manufacture and retail business, provided a control group.

The restriction of the inquiry to ' pneumonia ' is artificial, and as became evident in the course of the animal experiments, does not take account of the mode of action of manganese. On the other hand, diagnoses such as ' bronchitis ' are too vague for statistical purposes. For example, in 1944 no cases of ' pneumonia ' in manganese workers were recorded but 2 cases of bronchitis, stated by their medical attendants to be ' very severe,' occurred.

Clinical Features

The commonest complaint amongst workers is ' bronchitis,' a term which covers changes varying from nasopharyngitis to bronchitis and bronchopneumonia. People unused to exposure to manganese dust notice an irritation of the nose shortly after exposure: some hours later the nose feels blocked and epistaxis may occur. During the development of the animal chamber and of the method of creating dust-laden air, inadequate precautions were taken to enclose the dust, and two persons (T. A. L. D. and E. W.) concerned in the investigation suffered repeated colds and epistaxis, culminating in attacks lasting two or three days, which were thought to be influenza. When the association was recognized the use of adequate dust filters immediately stopped the attacks. This observation is of interest as the men working on the plant have for many years been provided with ' influenza mixture ' * which they imbibe liberally and with great faith when coming off work. The inhalation of potassium permanganate dust and permanganate spray is especially irritating to the nasopharynx, giving rise to coughing and retching. Men working on the plant become immune to the above symptoms.

Clinical examination of the respiratory system is unequivocal. A chronic granular pharyngitis is almost universal but cannot be said to occur more frequently than in other chemical workers. Foul teeth are very common and tracheitis is common. Except in men who have developed bronchitis, asthma or pneumonia, examination of the chest shows nothing abnormal.

' *Pneumonia.*'—So far as can be determined, all cases were diagnosed as lobar or bronchopneumonia, and no special points were noticed by the sufferers' private medical adviser. No special features were noticed except that the temperature and general condition of the patient responded

* A mixture containing ammonium acetate, camphor, squill and ether.

TABLE 1

THE INCIDENCE OF ILLNESS DIAGNOSED AS PNEUMONIA

	Manganese plant				Boots Health Insurance Society. Males 21–65 years			England and Wales †		U.S. Public Health Report, Washington §
	Maximum number employees (at end of any quarter)	Average monthly output (units)	Number cases pneumonia	Pneumonia rate per 1000	Number of male members	Number first certificate of pneumonia	Pneumonia rate per 1000	Number reported cases of pneumonia	Pneumonia rate per 1000 ‡	Pneumonia rate per 1000 males
1936	23		Records		5580	4	0·72			
1937	53	19·7	destroyed		5763	1	0·19			
1938	47	31·11	3	63	5991	3	0·52			
1939	40	39·9	2	50	6342	3	0·47	42,312	1·0	
1940	67	53·9	1	15	6586	6	0·91	47,875	1·2	
1941	101	79·19	3	30	6678	4	0·59	50,942	1·2	3·8
1942	124	97·7	3	24	6751	6	0·89			5·5
1943	101	91·16	2	20	4899*	6	1·20*			
1944	70	86·1	—	—	5008*	5	1·00*			
1945	75	81·19	2	26	4645*	1	0·02*			

* Civilians only; the pneumonia rate for these years is probably artificially high owing to removal of young and healthy males.
† Summary Report by the Ministry of Health, 1942. Appendix II, H.M.S.O. (including women and children).
‡ Population (estimated mid-year 1939) 41,460,000.
§ Public Health Report, 1943. Washington, **58**, 677.

FIG. 1.—Melted caustic potash (400°C.) is poured into manganese and lime and stirred: a violent reaction ensues in which large volumes of steam and quantities of dust are emitted.

FIG. 2.—Filling rotary kilns.

FIG. 3.—Discharge end of rotary kilns. Note enclosure and dust extraction.

FIG. 4.—Electrolysis to form potassium permanganate in caustic solution.

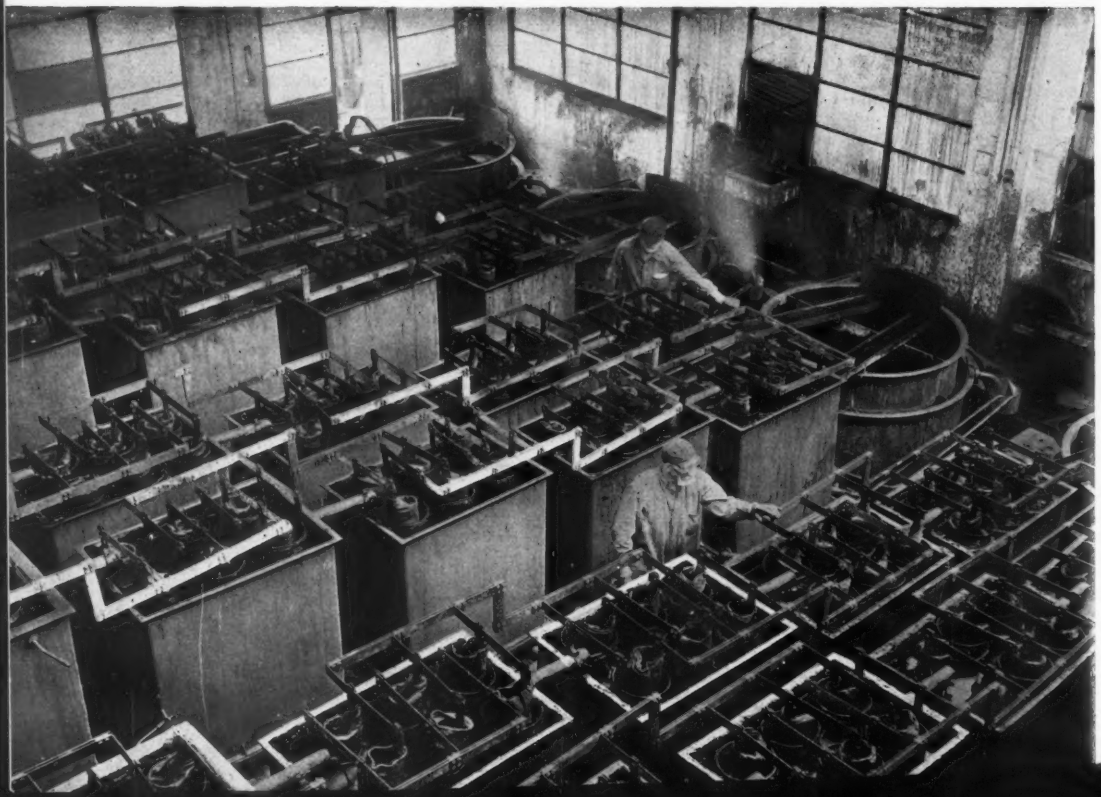


FIG. 5.—Charging roaster to dry recovered manganese dioxide.

FIG. 6.—Protective clothing to guard against caustic splashes and the inhalation of dust.

more slowly than usual to sulphonamides. The possible causal relationship to manganese was not suspected until subsequent enquiry.

Case No. 1. A male, aged 48 years, employed in the manufacture of potassium permanganate for ten years (previous employment general labourer and shop assistant), was seen during an attack by the kindness of Dr. R. Davidson. Clinically he appeared to be suffering from a typical lobar pneumonia with a high temperature (105°) which responded to sulphonamides though more slowly than usual. The right lower lobe appeared to be consolidated and pneumococci were present in the sputum in large numbers. Two months later an x-ray of the chest showed slight generalized fibrosis but no consolidation or other evidence of an earlier pneumonia.

Case No. 2. A male process worker, aged 43 years, entered the building trade on leaving school and became a builder's labourer until 38 years of age when he became a chemical worker. He was first employed on general chemical work, including the manufacture of halazone. He claims to have suffered from bronchitis at frequent intervals and to have had pneumonia as an infant, though when examined 16 months prior to the present illness no abnormality was discovered in his chest or elsewhere except for septic teeth. For the past five years (with the exception of two intervals of 3 months) he has been employed on the manufacture of potassium permanganate and latterly in recovering manganese dioxide which has to be 'dug' out of a vat, dried and sieved. His present illness started suddenly with pain in the left side of the chest, a cough and shortness of breath. Three days later he was admitted to Nottingham General Hospital under the care of Dr. J. W. Scott.

On admission, temperature was 103°, pulse 120, respiration 24. His teeth were septic, tongue clean. Air entry was diminished and coarse râles were present over the left chest. Blood pressure was 150/116. Other systems were normal. Sputum examined on two occasions did not show tubercle bacilli. A diagnosis of pneumonia was made.

He was treated with sulphathiazole and penicillin (120,000 units daily for three days) but his condition improved more slowly than would have been expected (see Temperature Chart).

An x-ray of his chest four days after admission (fig. 8) showed consolidation of the left upper lobe. The remainder of the lung field had an appearance of 'fluffiness' similar to that seen with early or slight pulmonary oedema. The bronchial shadows appeared swollen and indefinite in outline; some consolidation was present in the right lower lobe.

Fourteen days later the consolidation was resolving and the radiographic appearance of the rest of the lung field was normal except for increased bronchial markings (fig. 9). Nine weeks after admission to hospital an x-ray of the chest showed increased lung markings within normal limits (fig. 10). At this time he was free from symptoms; slight oedema of his left leg was present, probably due to circulatory stasis. The cardiovascular system showed no abnormality; the urine was normal.

Of the 16 men who suffered from 'pneumonia' (see Table 1), 8 examined in August and September 1945 showed neither clinical nor radiographic changes in the lungs which might be definitely ascribed to the inhalation of manganese. One man examined 18 months after pneumonia showed radiographic evidence of pleural thickening at both apices. Otherwise, the x-ray films showed increased lung markings such as are commonly ascribed to chronic bronchitis.

Bronchitis.—Attacks of mild bronchitis are frequent; records prior to 1942 are defective owing to enemy action, but since then 3 cases of very severe bronchitis with a superimposed asthmatic element have resulted in prolonged disability.

Case No. 3.* A man who had previously been a miner for 26 years, after working in the potassium permanganate plant for 4 years, suffered from three attacks of 'bronchitis' within a period of 2 months. Two months after the last attack he was in bed for 3 weeks with an illness diagnosed as 'pneumonia.' After getting up for 2 weeks he endeavoured to return to work but when examined prior to restarting work he was noticed to be very short of breath, with obvious asthma, even when at rest. Temperature 98°, pulse 80, and moist crepitations and wheezy expiratory ronchi were present all over the chest. A purulent gingivitis was present. An x-ray of his chest showed increased lung markings.

He was provided with a light outdoor job but he remained short of breath (with wheezy ronchi present all over his chest) for 2 months, when his symptoms and chest signs gradually improved. One year later, at his own request, he returned to work in the potassium permanganate plant, but after 2 months' exposure, suffered a recurrence of severe bronchitis and asthma.

Case No. 4.* A man was employed in shovelling recovered manganese mud, consisting mostly of manganese dioxide, into a dryer. He developed a

* Details of previous occupational history and age are irrelevant but not reported to avoid identification.

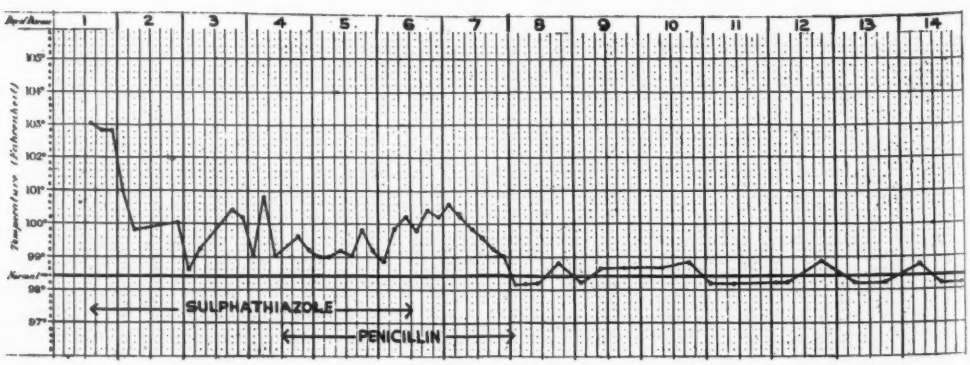


FIG. 7.—Temperature Chart of Case No. 2.

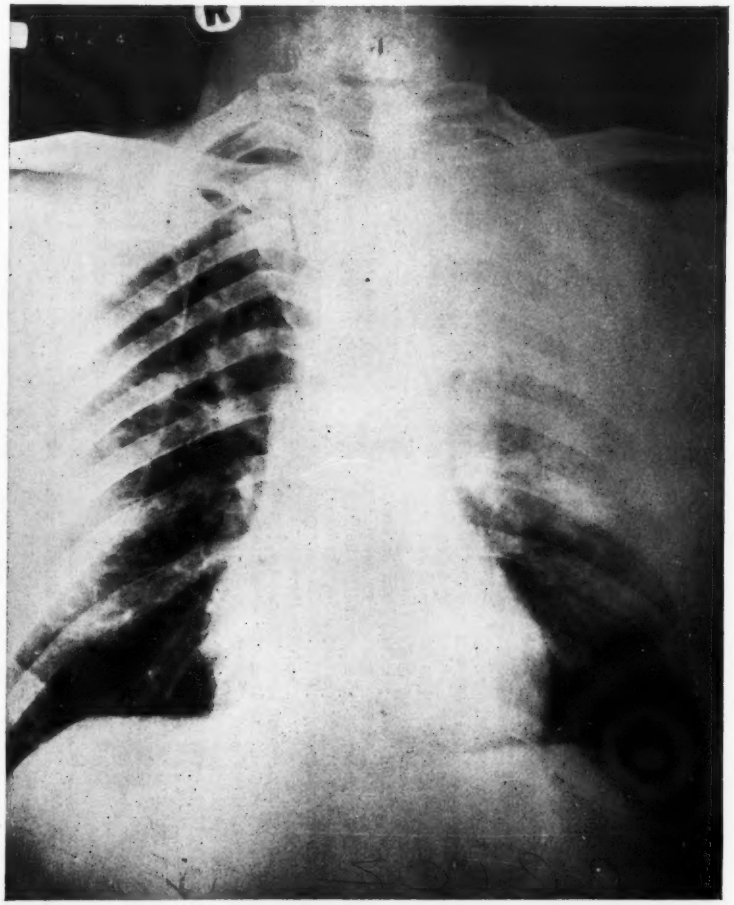


FIG. 8.—X-ray showing consolidation of left upper zone: some consolidation right base and indefinite outline of lung markings.

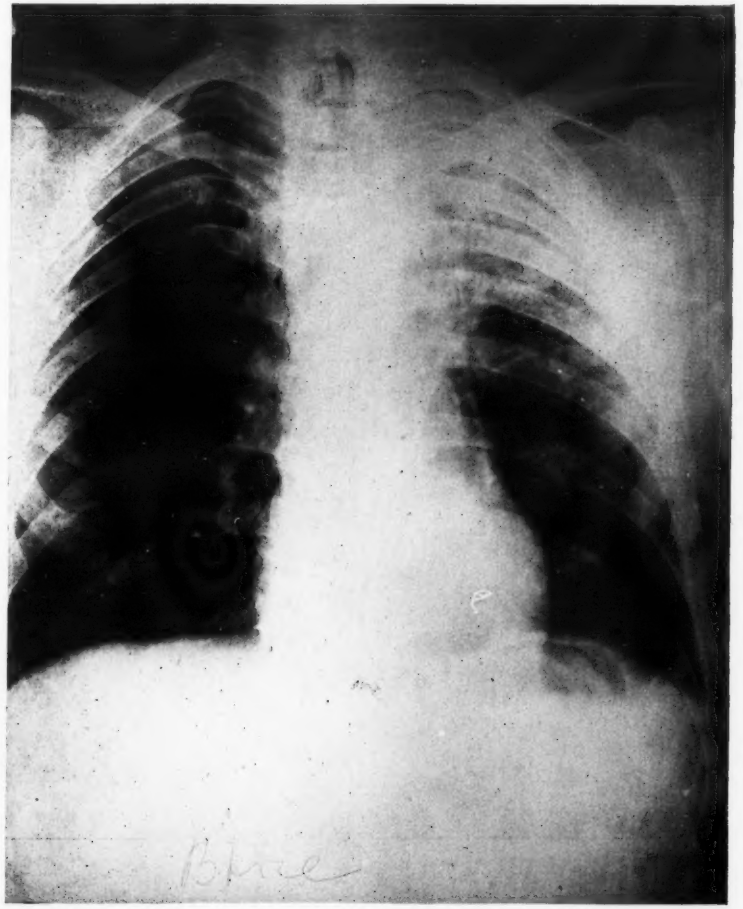


FIG. 9.—X-ray taken fourteen days later. Consolidation resolving: increased lung markings.

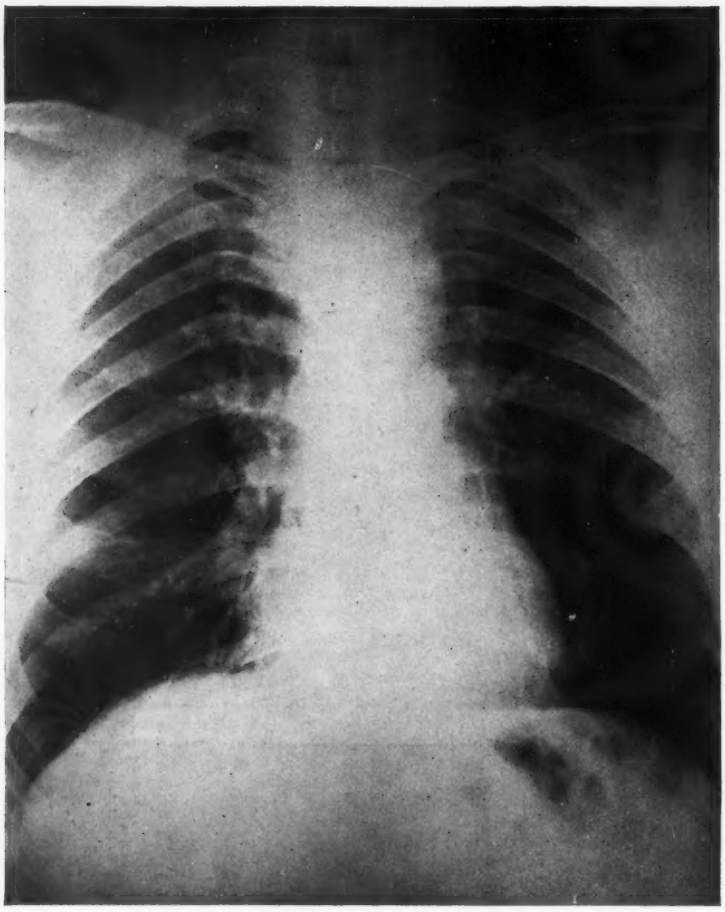


FIG. 10.—X-ray taken nine weeks later: normal appearance.

cough, and one week after starting this work the cough was severe, with much purulent sputum, and he was very short of breath even when at rest. Breathing was of the asthmatic type. In order to sleep he had to prop himself up into a semi-sitting position with pillows. Wheezy expiratory ronchi were present all over his chest. On removal from work his symptoms improved but recurred suddenly 2 weeks later on again inhaling manganese dust. An x-ray of his cheat showed increased markings at both bases with fluffy patches of consolidation. No tubercle bacilli were present in the sputum.

He continued to suffer from severe shortness of breath with attacks of asthma relieved by ephedrine for 3 months when he suffered a haematemesis due to a gastric ulcer. When he resumed work 8 months later, his chest symptoms were much improved, but 2 years after the original incident he is still liable to attacks of shortness of breath at night or on exertion. An x-ray of his chest showed increased bronchial shadows but no consolidation or fibrosis.

Seven other men besides those reported above, examined at various times because of absence due to chest symptoms, showed neither clinical nor radiographic abnormality on return to work. Three men who were examined as they had been employed for 9 years on the plant were free from symptoms and showed no abnormality on clinical and x-ray investigation.

Environmental Studies

The environmental studies are summarized in Tables 2 and 3. All dust counts were made with a thermal precipitator (Green and Watson, 1935).

The opinion that the dustiness of the plant varied with the weather is confirmed, dust counts being less when the wind is in the east. The particle size of the dust is small, approximately 80 per cent. of particles being less than 0·2μ in size, and nearly all particles less than 1μ. The mass concentration determined by the electrostatic precipitator bore no relation to the number of particles per cubic centimetre of air determined by the thermal precipitator or to the calculated mass concentration (Bedford and Warner, 1942).

TABLE 2

TABLE 2

ENVIRONMENTAL STUDIES

Date	Time	Site	Outside temp. ° F.	Wind direction	Speed m.p.h.	Climate	Plant temp. ° F.	Dust count: particles per c.c.	Differential count (per cent.)										
									0–·2μ	–·4μ	–·8μ	–1·2μ	–1·6μ	–2·0μ	–2·5μ	–3·0μ	–4·0μ	–5·0μ	Above 5μ
26.10.43	3.15 p.m.	1	56	S.W.	2–3	Fine	N.R.	8751	80·6	12·4	3·6	2·0	0·6	—	—	—	0·4	—	7μ, 0·2; 15μ, 0·2
21.12.43	11.30 a.m.	1	46	S.S.W.	17–20	—	N.R.	3654	75·25	9·75	6·0	3·25	1·5	1·5	1·0	1·0	0·5	0·25	—
1.11.44	2.55 a.m.	1	45	N.	4–7	Dull	N.R.	4740	84·4	10·1	2·1	1·4	0·3	0·3	0·1	0·3	0·1	0·5	7μ, 0·2; 10μ, 0·1; 15μ, 0·1
7.12.44	2.45 p.m.	1	36	S.W.	1–3	Frosty	45·0	2669	78·8	11·9	4·2	2·0	1·1	0·6	0·3	0·4	0·2	0·3	7μ, 0·1; 15μ, 0·1
7.12.44	3.7 p.m.	1	36	S.W.	1–3	Frosty	47·0	9019	86·5	9·3	2·2	0·7	0·2	0·1	0·3	0·1	0·4	0·1	7μ, 0·1
26.10.43	4.15 p.m.	2	56	S.W.	2–3	Fine	N.R.	9360	79·8	13·0	3·2	1·4	0·6	0·4	0·6	0·2	0·4	0·4	—
16.12.43	2.50 a.m.	2	39	E.	10–15	Misty	N.R.	4068	74·2	16·4	5·0	1·4	1·6	0·2	0·2	—	—	0·4	7μ, 0·2; 22μ, 0·2; 30μ, 0·2
2.11.43	10.15 a.m.	3	56	N.E.	3–6	Drizzle	N.R.	4811	83·9	12·3	2·7	0·5	0·3	0·3	—	—	—	—	—
12.1.44	11.45 a.m.	3	54	S.	Slight breeze	Mist	N.R.	8136	87·2	9·2	2·2	0·4	—	—	0·2	—	0·4	0·2	15μ, 0·2
18.1.44	—	3	52	S.W.	7–15	—	N.R.	19496	92·6	5·4	1·4	0·2	0·4	—	—	—	—	—	—
27.10.43	4 p.m.	5	59	S.W.	10–15	Sunny	67–68	1617	78·4	10·8	4·4	1·6	1·0	1·0	0·8	—	1·2	0·6	7μ, 0·2
15.12.43	2.35 a.m.	5	27	N.E.	5–10	Misty	51	7583	81·0	12·0	3·6	1·8	0·6	0·6	0·2	0·2	—	—	—
29.10.43	2.30 p.m.	6	56	S.E.	4–6	Fine	65·2	2653	87·6	10·2	1·4	0·6	—	0·2	—	—	—	—	—
14.12.43	4.5 a.m.	6	26	—	—	Fog	43·0	15143	80·0	13·6	4·8	1·4	0·2	—	—	—	—	—	—
2.11.43	11.45 a.m.	7	58	N.E.	3–6	Drizzle	65·0	6275	72·6	17·4	4·6	1·6	0·4	0·2	0·4	0·4	0·8	0·6	7μ, 0·4; 50μ, 0·4; 55μ, 0·2
15.12.43	7.13 a.m.	7	27	N.E.	5–10	Misty	53·0	8975	85·2	10·8	2·4	0·6	0·2	0·4	0·2	0·2	—	—	—
29.10.43	4.30 p.m.	8	55	E.	4–6	Fine	68·0	4060	92·5	5·6	0·9	0·5	0·2	0·1	0·1	—	—	0·1	—
14.12.43	4.40 a.m.	8	26	—	—	Fog	45·5	13503	81·6	12·4	4·2	1·4	0·4	—	—	—	—	—	—
18.1.44	—	8	—	S.W.	7–15	—	—	5169	82·4	12·0	3·4	0·8	0·6	0·4	—	—	0·2	0·2	—
29.10.43	10.40 a.m.	9	52	S.S.E.	2–6	Dull and misty	N.R.	11668	92·0	3·8	1·2	0·8	0·6	0·4	0·6	—	0·4	0·2	—
16.12.43	1.59 a.m.	9	39	E.	10–15	Misty	N.R.	8352	64·0	24·4	5·6	2·2	1·0	0·6	0·4	0·2	0·4	0·4	7μ, 0·2; 13μ, 0·2; 15μ, 0·2; 17μ, 0·2
1.11.43	3.30 p.m.	10	56	N.E.	3–6	Raining	N.R.	5438	81·6	13·4	3·0	1·4	0·2	—	0·4	—	—	—	—
15.12.43	5.03 a.m.	10	27	N.E.	5–10	Misty	N.R.	5268	79·0	13·4	4·2	2·0	0·8	—	—	0·4	0·2	—	—
4.11.43	11.45 a.m.	11	53	Still	—	Fog	N.R.	4119	72·0	18·8	5·2	3·2	0·6	—	—	—	—	—	7μ, 0·2
28.10.43	10.20 a.m.	12	52	S.W.	2–3	Fog	N.R.	8510	78·6	10·8	2·6	1·4	1·4	2·2	0·8	0·6	0·8	0·4	7μ, 0·2; 10μ, 0·2
16.12.43	3.05 a.m.	12	39	E	10–15	Misty	N.R.	2085	69·0	22·0	4·6	2·2	0·6	0·6	—	—	0·2	0·4	18μ, 0·2; 100μ, 0·2

TABLE 2—*continued*

ENVIRONMENTAL STUDIES—*continued*

Date	Time	Site	Outside temp. °F.	Wind direction	Speed m.p.h.	Climate	Plant temp. °F.	Dust count: particles per c.c.	Differential count (per cent. 0–·2μ	·–·4μ	·–·8μ	–1·2μ	–1·6μ	–2·0μ	–2·5μ	–3·0μ	–4·0μ	–5·0μ	Above 5μ
28.10.43	2.45 p.m.	13	56	S.W.	2–3	Misty	66·0	7589	78·0	12·6	3·8	2·4	0·6	0·8	0·2	0·2	0·6	0·6	40μ, 0·2
14.12.43	3.12 a.m.	13	26	Still	—	Fog	53·5	15098	78·2	13·0	6·0	2·0	0·6	0·2	—	—	—	—	—
21.10.43	11.20 a.m.	14	62	S.	15–20	Showery	84·0	2015	—	—	—	—	—	—	—	—	—	—	—
21.10.43	3.0 p.m.	15	61	S.	15–20	Showery	66·4	953	—	—	—	—	—	—	—	—	—	—	—
15.12.43	3.50 a.m.	15	27	N.E.	5–10	Misty	62·0	9389	84·6	9·4	4·4	1·2	0·4	—	—	—	—	—	—
14.12.43	7.30 a.m.	16	26	Still	—	Fog	52·0	8346	73·6	11·6	7·4	2·4	1·2	0·8	0·2	0·2	—	0·8	7·5μ, 0·2; 10μ, 1·0; 15μ, 0·2; 22μ, 0·2; 34μ, 0·2
15.12.43	2.55 a.m.	16	27	N.E.	5–10	Misty	55·0	7392	70·4	19·0	5·2	2·0	1·6	0·6	0·2	—	—	0·4	50μ, 0·2; 67μ, 0·2; 75μ, 0·2
1.11.44	11.30 a.m.	16	45	N.	4–7	Dull	—	5008	77·4	13·1	4·4	2·1	0·5	0·6	0·4	0·4	0·3	0·3	7μ, 0·2; 10μ, 0·1; 20μ, 0·1
7.12.44	11.10 a.m.	18	36	S.W.	1–3	Frosty	58·0	4826	75·7	16·0	2·5	1·0	0·7	0·7	0·6	0·8	0·5	0·6	7μ, 0·5; 10μ, 0·3; 15μ, 0·1
7.12.44	11.30 a.m.	18	36	S.W.	1–3	Frosty	58·0	3361	66·0	12·7	5·0	3·8	1·6	1·1	0·8	1·7	2·1	1·9	10μ, 1·5; 15μ, 0·8; 20μ, 0·5; 25μ, 0·1; 40μ, 0·2; 70μ, 0·1; 300μ, 0·1
26.10.43	11.50 a.m.	19	48	S.W.	2–3	Fine	56·0	31830	86·0	10·8	2·0	0·2	0·4	—	0·2	—	—	—	25μ, 0·2; 50μ, 0·2
15.12.43	1.47 a.m.	19	27	N.E.	5–10	Misty	41·5	9064	79·6	11·4	6·0	1·4	1·0	0·2	—	0·2	0·2	—	—
25.10.43	12.20 p.m.	20	50	S.W.	2–3	Fine	60–62	14995	71.8	23·6	3·5	0·4	0·5	0·1	—	0·1	—	—	—
14.12.43	1.55 a.m.	20	26	—	—	Fog	45·5	22903	79·6	12·4	5·2	2·0	0·4	0·2	—	0·2	—	—	—
1.11.44	5 p.m.	20	45	N.	4–7	Dull	—	7908	87·8	9·5	1·4	0·5	0·2	0·1	0·1	—	0·1	0·1	10μ, 0·1; 15μ, 0·1
7.12.44	12.10 p.m.	20	36	S.W.	1–3	Frosty	58·0	5937	87·8	8·6	1·7	0·8	0·3	0·1	—	—	0·1	0·3	10μ, 0·3
26.10.43	11 a.m.	21	36	S.W.	2–3	Fine	52·0	37040	84·6	11·6	2·0	0·4	0·2	—	—	0·2	0·4	—	7μ, 0·4; 10μ, 0·2
14.12.43	2.30 a.m.	21	26	Still	—	Fog	45·0	21439	82·4	11·4	2·8	0·6	1·2	0·4	0·2	0·2	0·2	0·2	7·5μ, 0·2; 10μ, 0·2
22.10.43	9.50 a.m.	22	52	S.	10–15	Fine	84·2	1850	82·8	14·7	1·5	0·3	0·3	0·2	—	—	0·1	0·1	—
16.12.43	3.43 a.m.	22	39	E.	10–15	—	50–52	2843	76·4	13·8	4·0	1·2	1·0	0·4	0·2	0·4	0·2	1·0	7μ, 0·2; 10μ, 0·2; 20μ, 0·2; 22μ, 0·2; 50μ, 0·4; 100μ, 0·2
22.10.43	12.10 p.m.	23	59	S.	10–15	Fine	79–81	8652	88·4	9·0	0·8	0·6	0·4	0·2	0·2	—	0·2	—	10μ, 0·2
16.12.43	4.15 a.m.	23	39	E.	10–15	—	57–59	5965	83·0	11·0	3·8	1·2	0·6	0·2	0·2	—	—	—	—

TABLE 3

ANALYSIS OF DUST (COLLECTED BY ELECTROSTATIC PRECIPITATOR) AND COMPARISON WITH THERMAL PRECIPITATOR DUST COUNTS

Date	Site	Thermal precipitator											Calculated mass concentration: mg./cu. m. as MnO_2. Sp.gr. 3·0		Electrostatic precipitator. Mass concentration: mg./cu. m.		Analysis
		No. particles per c.c.	Differential count (per cent.)									Above 5μ	All particles	Particles 1–5μ		Mn as MnO_2 per cent.	
			0–·2μ	–·4μ	–·8μ	–1·2μ	–1·6μ	–2·0μ	–2·5μ	–3·0μ	–4·0μ	–5·0μ					
1.11.44	1	4740	84·4	10·1	2·1	1·4	0·3	0·3	0·1	0·3	0·1	0·5	7μ, 0·2; 10μ, 0·1; 15μ, 0·1	40·2	4·5	0·7	43	
7.12.44	1	2669	78·8	11·9	4·2	2·0	1·1	0·6	0·3	0·4	0·2	0·3	7μ, 0·1; 15μ, 0·1	17·1	2·5	7·9*	66	Other major constituents Ca and K.
7.12.44	1	9019	86·5	9·3	2·2	0·7	0·2	0·1	0·3	0·1	0·4	0·1	7μ, 0·1	9·6	4·5			
1.11.44	16	5008	77·4	13·1	4·4	2·1	0·5	0·6	0·4	0·4	0·3	0·3	7μ, 0·3; 10μ, 0·1; 20μ, 0·1	83·4	4·5	37·3	58	Ba and Na present to extent of 1 and 0·1 per cent. respectively. Smaller traces of Si, Fe, Li.
7.12.44	18	4825	75·7	16·0	2·5	1·0	0·7	0·7	0·6	0·8	0·5	0·6	7μ, 0·5; 10μ, 0·3; 15μ, 0·1	68·4	7·8	38·3*	54	
7.12.44	18	3361	66·0	12·7	5·0	3·8	1·6	1·1	0·8	1·7	2·1	1·9	10μ, 1·5; 15μ, 0·8; 20μ, 0·5; 25μ, 0·1; 40μ, 0·2; 70μ, 0·1; 300μ, 0·1	—	16·8			
1.11.44	20	7908	87·8	9·5	1·4	0·5	0·2	0·1	0·1	—	0·1	0·1	10μ, 0·1; 15μ, 0·1	53·4	1·8	0·42	41	
7.12.44	20	5936	87·8	8·6	1·7	0·8	0·3	0·1	—	—	0·1	0·3	10μ, 0·3	29·7	3·0	9·2	65	

* Two thermal precipitator counts during one run of electrostatic precipitator.

TABLE 4
STANDARDIZATION OF APPARATUS
(All air flows 4 litres over dust and 6 litres through by-pass)

Experiment No.	Manganese dust: Dust count particles per c.c.	Interval after preceding count	Differential count (per cent.)										
			0–·2μ	–·4μ	–·8μ	–1·2μ	–1·6μ	–2·0μ	–2·5μ	–3·0μ	–4·0μ	–5·0μ	Above 5μ
Control	No strip												
1	2552		62·9	13·8	9·0	5·0	3·0	1·2	0·6	1·5	1·2	1·0	7μ, 0·6; 10μ, 0·1; 15μ, 0·1
2	2569	Consecutive	65·8	16·8	8·0	3·4	1·2	1·0	0·6	0·6	0·8	0·7	7μ, 0·5; 10μ, 0·4; 15μ, 0·1; 50μ, 0·1
3	2199	10 minutes	65·1	15·3	8·5	3·6	2·0	1·2	0·8	0·9	1·2	0·5	7μ, 0·4; 10μ, 0·3; 15μ, 0·1; 20μ, 0·1
4	2125	15 minutes	70·8	14·3	8·6	2·7	0·9	0·7	0·4	0·6	0·5	0·2	10μ, 0·1; 15μ, 0·1; 50μ, 0·1
Control	No strip												
5	1663		58·8	17·6	9·6	4·4	3·4	1·0	0·7	1·3	1·1	1·2	7μ, 0·3; 10μ, 0·4; 15μ, 0·1; 20μ, 0·1
6	1401	5 minutes	66·6	14·7	8·0	4·0	2·6	0·7	0·6	0·5	0·6	0·7	7μ, 0·6; 10μ, 0·2; 15μ, 0·2
7	1207	5 minutes	60·3	15·6	9·4	6·0	2·0	1·6	0·7	1·4	0·8	1·3	7μ, 0·6; 10μ, 0·1; 15μ, 0·1; 22μ, 0·1
Control	No strip												
8	1629		74·4	13·0	4·5	2·4	1·0	0·8	0·6	1·1	1·1	0·6	7μ, 0·3; 10μ, 0·2
9	1891	2½ hours	72·3	17·4	5·0	1·9	0·8	0·9	0·6	0·5	0·4	0·1	7μ, 0·1
10	1948	Air off for 16 hours	46·1	20·7	10·6	5·4	5·1	4·1	2·0	1·4	1·9	1·5	7μ, 0·8; 10μ, 0·4
11	1715	5 hours	51·9	23·1	9·6	5·8	2·6	1·5	1·2	0·8	1·6	1·1	7μ, 0·5; 10μ, 0·3
Control	No strip												
12	2689		32·6	22·9	17·6	8·0	4·7	3·9	2·3	2·7	1·7	1·7	7μ, 0·9; 10μ, 0·6; 15μ, 0·3; 20μ, 0·1
13	1570	3 hours	39·7	24·5	14·0	6·5	4·6	2·9	1·0	2·0	2·0	1·6	7μ, 0·45; 10μ, 0·3; 15μ, 0·3; 50μ, 0·15
	Foundry sand												
Control	No strip												
14	1150		26·5	11·5	13·0	6·5	9·0	4·5	2·0	5·0	6·5	8·0	7μ, 3·5; 10μ, 1·0; 15μ, 2·0; 20μ, 1·0
15	1133	5 minutes	51·2	10·2	7·4	4·7	3·5	3·7	2·2	4·1	4·2	4·7	7μ, 2·0; 11μ, 1·1; 15μ, 0·6; 20μ, 0·1; 22μ, 0·1; 25μ, 0·2
16	1709	10 minutes	48·2	17·1	10·7	6·6	3·6	2·8	2·1	2·2	2·1	2·3	7μ, 1·3; 10μ, 0·5; 15μ, 0·3; 20μ, 0·1; 22μ, 0·1
Control	No strip												
17	1825		33·2	11·6	10·8	9·5	7·3	4·7	4·0	5·8	4·7	4·1	7μ, 2·6; 10μ, 1·1; 15μ, 0·6
18	1658	2 hours 10 minutes	20·7	9·3	13·5	10·4	9·4	7·0	4·3	6·9	8·1	5·4	7μ, 3·2; 10μ, 1·2; 15μ, 0·3; 20μ, 0·1; 22μ, 0·1; 25μ, 0·1
19	1914	30 minutes	47·7	11·8	11·5	6·8	4·7	3·6	2·2	3·3	3·2	2·3	7μ, 1·6; 10μ, 1·0; 15μ, 0·3
20	2917	30 minutes	32·1	8·8	10·4	8·3	7·1	5·9	3·8	6·2	6·1	5·0	7μ, 3·6; 10μ, 1·3; 15μ, 1·0; 20μ, 0·1; 22μ, 0·2; 30μ, 0·1
21	3213	55 minutes	49·2	15·0	8·0	4·8	3·7	3·7	2·6	3·6	3·7	3·3	7μ, 1·1; 10μ, 0·5; 15μ, 0·5; 22μ, 0·2; 25μ, 0·1
22	2370	35 minutes	62·0	15·6	9·2	3·6	2·6	1·8	1·0	1·4	0·8	1·0	7μ, 0·4; 10μ, 0·6
23	3737	30 minutes	59·6	17·4	7·4	3·2	3·6	1·6	2·2	1·6	1·6	1·0	7μ, 0·8
Control	No strip												
24	1988												
25	1561	4 hours 15 minutes	33·8	15·2	8·6	7·8	5·4	5·2	5·4	6·4	4·6	5·0	7μ, 1·2; 10μ, 0·6; 15μ, 0·6; 22μ, 0·2

Samples of dust for analysis were obtained with an electrostatic precipitator. The constitution of the dust varies in different parts of the plant. In the mill room the dust consists of manganese dioxide, but elsewhere, though the major constituent is manganese dioxide, manganite, manganate and permanganate of potassium may also be present. These substances would be rapidly reduced by body fluids to manganese dioxide. The manganese content of the dust, collected by the electrostatic precipitator, expressed as manganese dioxide varied from 41 to 66 per cent.

Other major constituents were potassium and calcium, the latter originating from the lime used in the reactions. Alkalinity of the dust collected from walls and ledges, calculated as potassium carbonate, varied from 1·01 per cent. in the mill room to 11·8 per cent. in the main hall. For the purpose of animal experiments dust was collected from the ledges and walls. (The absolute density of mixed samples was found to be 3·0.)

Since this investigation began, many improvements have been effected in working conditions, for example, exhaust ventilation has been fitted to the charging and discharging ends of the kilns (fig. 3).

II. Animal Experiments

Literature

Jötten and Reploh (1938) exposed rabbits to pyrolusite dust and produced broncho-pneumonia. The degree and severity of the broncho-pneumonia was increased by inoculation with pneumococci type II, either alone or accompanied by daily vigorous twisting of the animal. These authors do not report control experiments where animals not exposed to manganese dust were similarly inoculated.

Jötten, Reploh and Hageman (1939), discussing further experiments by Jötten (1939), described the histological changes in rabbits' lungs after exposure to manganese dust as a severe bronchitis with bronchiolitic changes. No signs of croupous pneumonia were present and these authors described the cellular reaction as a leucocytic and partly lymphocytic infiltration. Illustrations accompanying their paper show the large, apparently empty, mononuclear cells, characteristic of the present experiment. They also stated that changes could not be produced in mice's lungs unless a concomitant inhalation of pneumococci (either dead or alive) was employed. They concluded that manganese has a specific local action on the tissues.

Reploh (1943) has reported a series of experiments in which mice were exposed to various dusts including manganese carbonate dust from a Siemen-Martin furnace (manganese content 7–9 per cent.) and basic slag. With the exception of Siemen-Martin furnace dust, the mice failed to develop lung infections unless they were either previously treated with inhalations of dead pneumococci or previously treated in a similar way followed by injection with dead or living pneumococci after the exposure of dust.

Dust-feed Apparatus

Owing to the heavy nature of manganese dioxide and the clinging nature of dust collected from the walls and ledges of the plant, the method described by Argyll Campbell (1934) did not prove satisfactory for the reproduction of the plant atmosphere. The major disadvantage of other dust-feed apparatus is the large amount of air required and the need to keep the level of the dust constant in relation to the large volume of relatively slow-moving air. The mechanical arrangements to achieve this are bulky, not easily movable and complicated (Stead, Dernell and Nau, 1944). If a small volume of air at a relatively high velocity were employed, and a compressed air cylinder* used as the source, many of these objections would be overcome (figs. 12 and 13).

Air was blown from a jet on to the surface of the dust 6 cm. away from the jet (fig. 12). The resultant lowering in the level of the dust during the experiment was negligible in relation to the air speed and distance from the jet. The dust was stirred with a paddle which revolved four times a minute. If desired, the dust-laden air could be diluted with clean air so that the dust concentration of the air entering the animal chamber might be controlled.

A rectangular animal chamber 600 × 550 × 500 cm., similar to that described by Argyll Campbell (1934) was used. The dust count in the chamber was kept constant by adjusting the flow of air through the jet between 1½–4 litres per minute and the clean air between 8½–6 litres per minute so that a total flow of 10 litres per minute was maintained. Before estimating the dust concentration the apparatus was run for at least half an hour to displace the air from the animal chamber. Standardization was effected by placing the thermal precipitator in the animal chamber: for any given dust and air flow it was always possible to reproduce within practical limits the same dust concentration for periods up to 5 hours (Table 5). The dust container was refilled as necessary but would, at the above air flows, last for 3 or more hours. In order to prevent the temperature rising in the animal chamber during an experiment, a coil of copper tube ½ inch in diameter, making three circuits of the chamber, through which cold water was passed, proved very effective. For experiments with mice the chamber temperature was kept at 65° F. The whole apparatus was installed in a thermostatically controlled room (temperature 65° F.) and in which the mice were kept when out of the chamber.

Animal Experiments

I. Manganese Dust (from Main Hall)

Seventy-two mice each weighing between 25–30 gm. were divided into two groups: (*a*) 60 mice

* A small compressor may be substituted but an after-cooling apparatus and filter are essential.

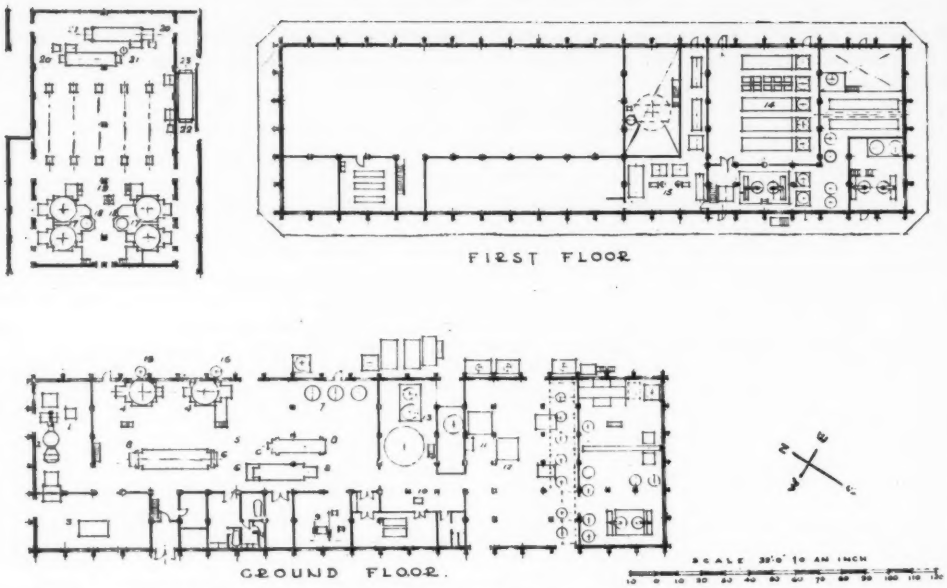


FIG. 11.—Plan of plant: figures refer to sites in which estimates of dust in atmosphere were performed.

1. Mill room near bags.
2. Mill room near switch gear.
3. Sieve room near sieve.
4. Caustic pots Nos. 1 and 2.
5. Main hall—general atmosphere centre.
6. Charge end kilns Nos. 1, 2 and 6.
7. Nos. 1, 2, 3 roasters near roaster.
8. Discharge and kilns Nos. 1, 2 and 6.
9. Roast crusher.
10. Paxtman mud discharge sump.
11. Charging trays permanganate.
12. Discharging trays permanganate.
13. Dissolver.
14. Cell room.
15. Paxtman filter room.
16. Reaction pots Nos. 1 and 2.
17. Caustic pots Nos. 3, 4, 5 and 6.
18. Reaction pots Nos. 3 and 4.
19. General atmosphere.
20. Charge end kilns Nos. 4 and 5.
21. Discharge end kilns Nos. 4 and 5.
22. Charge end kiln No. 3.
23. Discharge end kiln No. 3.
24. Unloading manganese ore on dock.

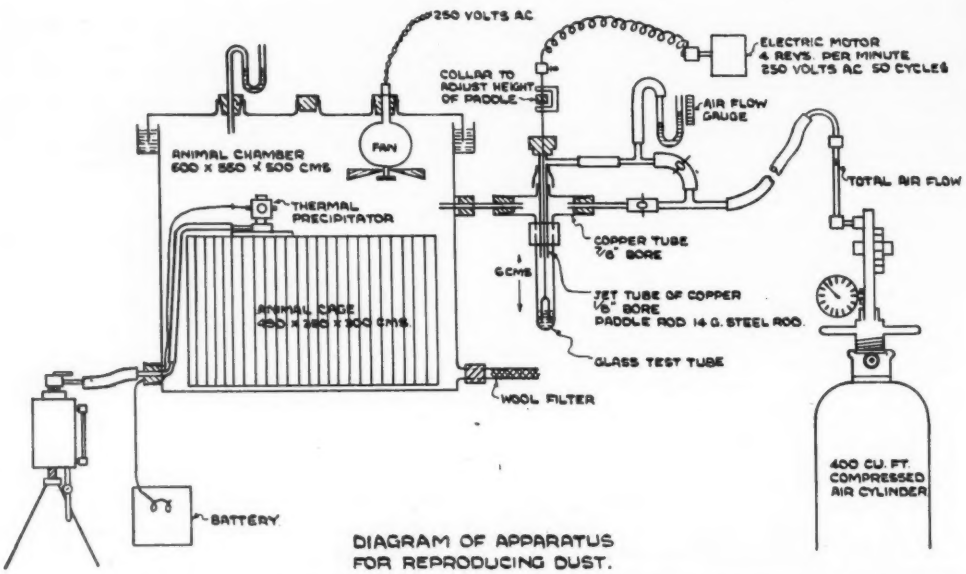


FIG. 12.—Diagram of dust-feed apparatus and animal chamber. Note cooling coil (see text) fitted inside chamber not shown in diagram.

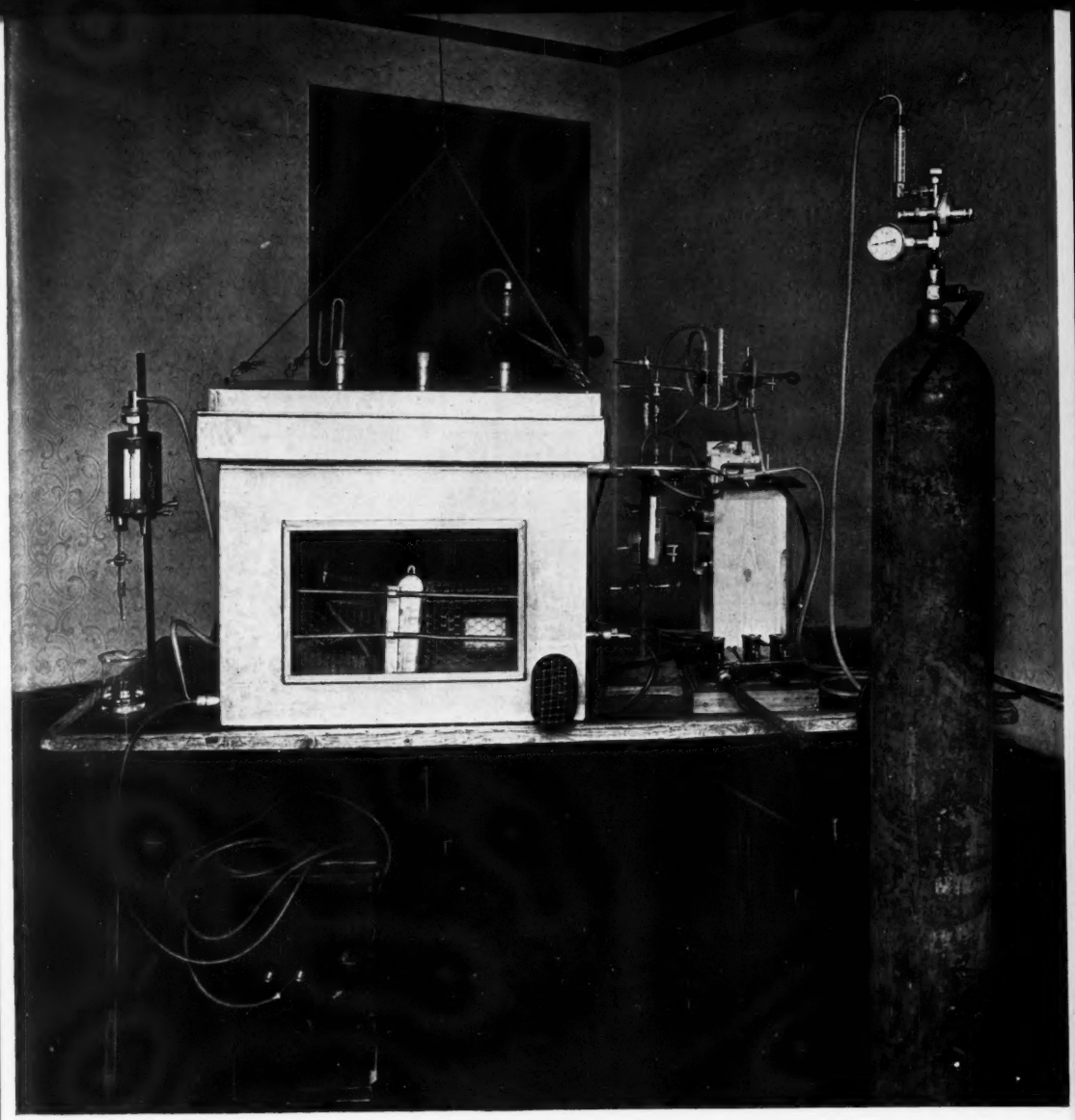


FIG. 13.—Photograph of the apparatus. Note cooling coil inside dust chamber and Mark IV respirator pad used as dust filter.

Day	Daily length of exposure (starting at 10 a.m. and 3 p.m.) minutes	Dust count: particles per c.c.	Remarks*
1	2×120	2529	1 dead. ? reason.
2	1×120	1747	
3	2×120	2364	
4	2×120	2786	
5	2×120	1441	1 dead. 1 blind—killed. *Lungs section:* intense infiltration with lymphocytes and large mononuclear cells especially around arteries and bronchi. 1 control dead.
6	2×120	940	
7	2×120	1658	3 dead.
8	1×120 and 1×60	2472	1 blind and ill—killed. *Lungs section:* few small patches of mononuclear infiltration especially subpleural, and peribronchial. TOTAL died or killed 7 and 1 control.
9	No exposure.	—	1 dead. *Lungs section:* alveoli appear large; patches of consolidation and extensive generalized interstitial mononuclear infiltration; exudate in alveoli.
10	No exposure.	—	
11	No exposure.	—	1 dead. TOTAL died or killed 2.
	Mice made up to original number.		
12	1×30 and 1×120	1760	
13	1×120	1749	1 dead. *P.M.* Nares obstructed with dust; stomach full of manganese dust. Lungs broncho-pneumonia. Spleen small with area of peritoneal thickening. *Section:* spleen nothing abnormal; lungs subpleural and peribronchial infiltration, patchy and intense. Stomach normal.
14	2×120	—	
15	2×120	2387	
16	No exposure.	—	1 dead.
17	2×120	2535	1 dead.
18	1×120 and 1×70	923	
19	2×70	2392	1 dead. *Lungs section:* exudate in bronchi with dust-laden cells in bronchi, alveoli and interstitial spaces; intense peribronchial infiltration; small haemorrhagic areas.
20	2×70	1925	
21	2×70	—	1 ill—killed. *P.M.* Nares and stomach full of dust, ? broncho-pneumonia. *Lungs section:* slight infiltration; collection of large mononuclear cells around bronchi and arteries. Oedema of the lungs.
22	No exposure.	—	1 dead.
23	2×70	—	1 ill—killed.
24	2×70	1606	1 dead.
25	2×70	763†	
26	2×70	—	2 ill—killed. (1) *P.M.* Liver mottled, nil else apparent. *Lungs section:* interstitial mononuclear infiltration. (2) *P.M.* Large blood clot filling whole of pleural cavity; gross broncho-pneumonia. *Lungs section:* subpleural haemorrhage, generalized infiltration and patches of intense large mononuclear consolidation; some polymorphs seen; mediastinal glands showed heavy round cell infiltration. Bronchial epithelium swollen; exudate in bronchi and alveoli.
27	2×70	—	2 ill-killed. (1) *P.M.* Nothing abnormal. *Lungs section:* aggregates of dust cells in interstices of lungs. Mononuclear infiltration around bronchi and areas of consolidation and necrosis. (2) Paralysis of hind leg. *P.M.* Nothing abnormal. *Lungs section:* intense infiltration around arteries and bronchi with areas of consolidation. Brain ? round cell infiltration.
28	No exposure.	—	3 dead. *P.M.* Nothing abnormal. *Lungs section:* intense peribronchial infiltration especially of large mononuclear phagocytic cells; few lymphocytes and some polymorphs. Swelling of bronchial and alveolar epithelial cells. (See Fig. 14, B.) Brain ? patches of round cell infiltration.
29	No exposure,	—	1 dead. TOTAL dead or killed 16.

* Where no post-mortem examination has been made of dead mice this is because the time of death (for example, week-end) lead to autolysis, or because of cannibalism.

† One or more mice escaped from cage and interfered with thermal precipitator.

TABLE 5

NUMBER OF MICE ALIVE

Days after infection	Mice treated with manganese: 20 mice		Normal mice+pneumococci		Manganese-treated mice+pneumococci		Controls	
			Susp. A	Susp. B	Susp. A	Susp. B	Normal mice	Normal mice from manganese room
0	10	10	10	10	10	10	10	8
1	10	10	10	10	7	10	10	8
2	10	10	1	0	0	0	10	8
3	10	9	1	0	0	0	10	8
4	9	9	0	0	0	0	9	8
5	9	9	0	0	0	0	9	8
6	9	9	0	0	0	0	9	8

(in two cages of 30 each) which were exposed to dust, (*b*) 12 mice as controls, which except for exposure to dust were kept and treated in an identical manner. The dust contained 70 per cent. of MnO_2 and its alkalinity, mostly present in the form of potassium carbonate, calculated as KOH was 9·6 per cent., or calculated as K_2CO_3 was 11·8 per cent.

Exposures to dust for 2 hours twice daily were given 5 days a week for 14 exposures but the mice appeared ill and 7 exposed mice died or were killed. One control mouse died. After an interval of 8 days (during which 3 mice died) the mice were made up to the original number and exposed twice daily, but after 10 exposures the time of exposure was reduced to 70 minutes twice daily, 5 days a week, for 26 exposures (during which 16 mice died or were killed). Dust counts inside the chamber were performed every second or third exposure.

Bacteriological Results.—At the end of this time the exposed mice, though not obviously ill, were in poor condition and less well than the control mice. The exposed mice had lost weight, 11 mice weighing 23–24 gm. (As this was an unexpected effect, individual weights had not been kept.) Four days after the last exposure the exposed mice were tested for susceptibility to pneumococci.

The mice were divided by random selection in six groups as shown in Table 5, and given intraperitoneal injections of pneumococcus (type II): suspension A, each mouse received 0·5 ml. of a suspension containing 240,000 organisms per ml., suspension B, each mouse received 0·5 ml. of a suspension containing 12,000 organisms per ml.

The doses had been carefully standardized but the suspension proved about five times as toxic as expected. Post-mortem smears and cultures from the lung, spleen and heart showed large numbers of pneumococci.

Nine of the manganese-treated control mice were infected with 0·5 ml. of a suspension of pneumococci containing 800 organisms per ml., the remaining 9 being kept as controls.

In view of the poor condition of the manganese-exposed mice the results were not considered significant and do not suggest that the inhalation of manganese increases the susceptibility to pneumococci.

TABLE 6

NUMBER OF MICE ALIVE

Time after infection	Manganese treated mice+pneumococci	Normal mice+pneumococci	Controls	
			Normal mice	Manganese treated mice
0 hours	9	9	9	9
24 ,,	9	9	9	9
48 ,,	4	8	9	9
54 ,,	1	3	9	9
72 ,,	1	0	9	9
4 days	1	0	9	9

Absorption of Manganese.—Four days after the end of exposure, 3 exposed mice and 3 control mice were killed. The lungs and brains were removed and grouped and 1 mouse of each group skinned. After wet oxidation, the total manganese content was estimated spectrographically, using the cathode layer D.C. method.

	Exposed mice		Control mice	
	Total of 3 mice	Average per mouse	Total of 3 mice	Average per mouse
	μgm.	μgm.	μgm.	μgm.
Brains ..	1·5	0·5	1·3	0·4
Lungs ..	1·5	0·5	0·5	0·17

II. Calcium Carbonate Dust (Control Experiment)

Heavy calcium carbonate (s.g. 2·9) was used as a non-toxic dust of similar specific gravity and particle size.

Seventy-two mice each weighing between 25–30 gm. were divided as in Experiment I.

Exposures to dust for 70 minutes twice daily were given 5 days a week for 30 exposures. Dust counts inside the chamber were performed every second or third exposure. No control mice died.

Day	Daily length of exposure (starting at 9.30 a.m. and 2.30 p.m. minutes	Dust count: particles per c.c.	Remarks
1	2×70	991	
2	2×70	—	
3	2×70	—	
4	2×70	1629	
5	2×70	—	
6	2×70	—	
7	2×70	1259	
8	2×70	1903	1 dead.
9	2×70	—	
10	2×70	1675	
11	2×70*	1025	1 dead. Lost 6 gm. in weight. *Lungs section:* slight round cell infiltration and some subpleural haemorrhage.
12	2×70	—	1 dead. *Lungs section:* some subpleural mononuclear and lymphocytic infiltration.
13	—	—	
14	2×70	2045	2 dead.
15	2×70	1640	1 dead.
16	—	—	2 dead. 1 killed. (1) No P.M. (2) *P.M.* Lungs normal, section few dust cells and phagocytic cells. (3) Eye injury—killed. *Lungs section:* normal.
17	2×70	—	1 dead. *Lungs section:* small collection of polymorphs and lymphocytes; few dust cells.
	Exposures discontinued.		
18	—	—	1 dead.
19	—	—	1 dead. *Lungs section:* normal. (See Fig. 14, A.)
20	—	—	1 dead. No P.M.

* The mice were stained for identification purposes and 19 stained with brilliant green developed bald patches where they had been stained. These 19 mice were isolated on the 11th day together with 6 unstained mice and were not subjected to further exposures of dust. The remaining 33 mice were submitted to a further 8 exposures, making 30 exposures in all. Two unstained mice and one stained mouse died after cessation of exposure.

Weight.—Before exposure, 25–30 gm.

At end of experiment:

33 mice after 30 exposures average 29 gm. (range 21–32 gm.).

58 mice after 32 exposures average 27 gm. (range 21–31 gm.).

12 control mice average 32 gm. (range 28–35 gm.).

Bacteriological Results.—(1) Pneumococcus. The mice were divided as shown in Table 7 and injected intraperitoneally with pneumococcus (type II). Suspension A, 0·5 ml. of suspension of 110 organisms per ml., and suspension B, 0·5 ml. of suspension of 20 organisms per ml.

(II) Streptococci. A similar test was performed using *Streptococcus Haem.* (Richards) 0·5 ml. of a suspension of 245 organisms per ml. being given intraperitoneally (Table 8).

TABLE 8

NUMBER OF MICE ALIVE

Days after infection	Mice treated with $CaCO_3$	Mice treated with $CaCO_3$ +Streptococci	Normal mice +Streptococci	Normal mice Controls
0	11	10	10	8
1	11	10	9	8
2	11	2	2	8
3	11	2	2	8
4	11	2	1	8
5	11	2	1	8
6	11	2	1	8

The results show no significant difference in the rate of death of mice exposed to calcium carbonate dust and normal mice.

It was very noticeable that the mice exposed to manganese in Experiment I were in a worse condition than those exposed to calcium carbonate. The latter group, apart from some loss of weight, appeared to be of normal vigour but the manganese exposed mice, though not obviously ill, were undoubtedly made less well by exposure to manganese.

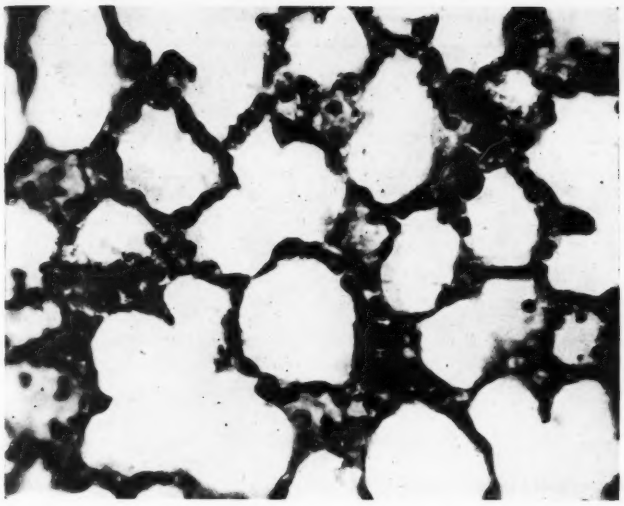
III. Manganese Dust (from Mill Room)

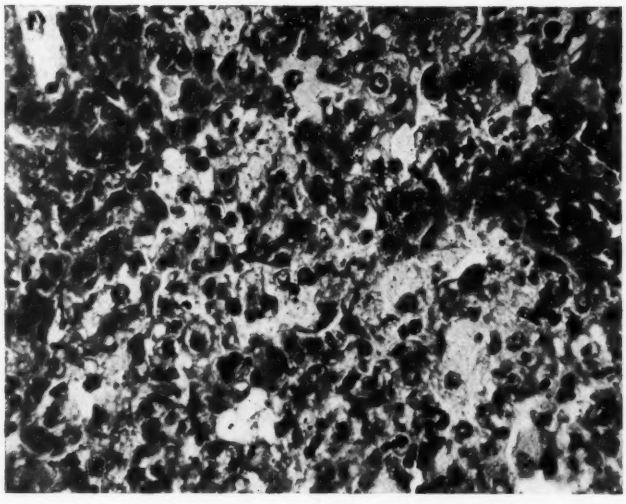
Seventy-two mice, each weighing between 25–30 gm., were divided into two groups: (*a*) 60 mice (in two cages of 30 each) which were exposed to dust for 34 exposures of 70 minutes (twice daily for 5 days a week), (*b*) 12 mice as controls. The dust contained 74 per cent. MnO_2 and its alkalinity calculated as KOH was 0·8 per cent. or calculated as K_2CO_3 was 1·01 per cent.

TABLE 7

NUMBER OF MICE ALIVE

Days after infection	Mice treated with calcium carbonate		Calcium carbonate-treated mice+pneumococci		Normal mice+pneumococci		Normal mice	Normal from $CaCO_3$ room
			Susp. A	Susp. B	Susp. A	Susp. B		
0	12	12	12	12	12	12	12	12
1	12	12	12	12	12	12	12	12
2	12	12	4	6	5	1	12	12
3	12	12	1	1	0	0	12	12
4	12	11	1	1	0	0	12	12
5	12	11	1	1			12	12
6	11	10	1	1			12	12
7	11	10	1	1			12	12





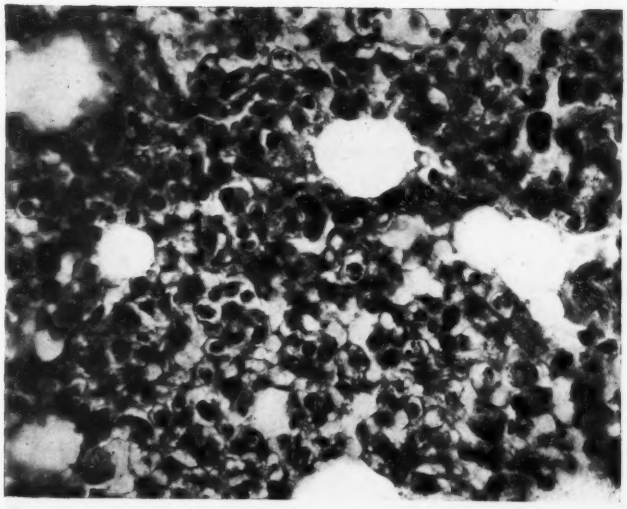


FIG. 14.

A. Section ×700 : lungs of mouse exposed to inhalation of calcium carbonate twice daily for 15 days. Normal lung structure.

B. Section ×700 : lungs of mouse exposed to manganese dust (MnO_2 content 70 per cent. alkalinity as K_2CO_3 11·8 per cent.) twice daily for 25 days. Structure almost unrecognizable due to intense infiltration of large mononuclear cells, many of which contain cellular debris and a few contain dust particles.

C. Section ×700 : lungs of mouse exposed to manganese dust (MnO_2 content 70 per cent. alkalinity as K_2CO_3 1·01 per cent.) twice daily for 19 days. Appearance is similar to B; intense infiltration with large mononuclear cells ; swelling of alveolar cells can be seen. Note absence of polymorphonuclear cells.

Day	Daily length of exposure (starting at 9.30 a.m. and 2.30 p.m.) minutes	Dust count: particles per c.c.	Remarks
1	2×70	1077	
2	2×70	1838	
			Range gm. / Average gm.
			Manganese-exposed mice weighed .. .. .. 25–29 / 26
			Control mice weighed .. .. 25–30 / 27
3	2×70	1242	
4	2×70	5412	
5	2×70	4136	
6	2×70	1559	
7	2×70	2261	
8	2×70	2643	
			Range gm. / Average gm.
			Manganese-exposed mice weighed .. .. .. 18–28 / 23
			Control mice weighed .. .. 21–28 / 24
9	—	—	1 dead.
10	2×70	2506	
11	1×70	1189	
12	1×70	2185	
13	2×70	1242	1 ill—killed. *P.M.* Subpleural haemorrhages. *Lungs section:* occasional dust cells, mononuclear infiltration; some lymphocytes and a few polymorphs.
14	2×70	2455	
15	2×70	—	
			Range gm. / Average gm.
			Manganese-exposed mice weighed .. .. .. 18–27 / 23
			Control mice weighed .. .. 18–30 / 25
16	—	—	1 dead.
17	2×70	1755	
18	2×70	—	1 dead. *P.M. Lungs:* few subpleural haemorrhages. *Section:* intense peribronchial mononuclear phagocytic infiltration, several dust cells, few lymphocytes and polymorphs. *Liver* engorged with numerous areas varying from pinhead to small pea, of yellow coloration all over surface and throughout section, probably necrosis or fatty infiltration. *Section:* areas of necrosis and early fibrosis, some interstitial haemorrhage.
19	2×70	—	1 dead.
20	2×70	—	1 dead. No *P.M.* Both lungs were collapsed. No haemorrhages, liver 'opaque.' *Lungs section:* mononuclear infiltration and haemorrhage. *Liver:* interstitial haemorrhage and early general necrosis.
	Exposure stopped.		
			Range gm. / Average gm.
			Manganese-exposed mice weighed .. .. .. 18–29 / 24
			Control mice weighed .. .. 18–31 / 25
	3 days after exposure.		1 dead.
			1 dead. *P.M. Lungs section:* large phagocytes loaded with dust in bronchi, alveoli and interstitial spaces. Numerous interstitial haemorrhages, no dust cells seen in lymphatic glands. Liver normal.
	4 days after exposure.		1 dead.
			1 dead. *P.M.* Haemorrhage in pleural cavity. *Lungs section:* intense phagocytic cell infiltration, few dust cells. Only occasional polymorphs or lymphocytes. (See fig. 10, C.) Liver, section normal.
	No control mice died.		

Bacteriological Results.—The mice were divided nto groups as shown, and injected intraperitoneally:

(1) Pneumococcus (type II). Each mouse received 0·5 ml. of a suspension containing 70 organisms/ml.

(2) Pneumococcus (type IV). Each mouse received 0·5 ml. of a suspension containing 8400 organisms/ml.

(3) *Streptococcus Haem.* Richards. Each mouse received 0·5 ml. of a suspension containing 200 organisms/ml.

TABLE 9

PNEUMOCOCCAL AND STREPTOCOCCAL INFECTIONS IN MANGANESE-TREATED MICE

Organisms	Pneumococcus, type II		Pneumococcus type IV		Strep. haem. (Richards)		Controls		
Number organisms injected	35		4200		100		—		
Days after infection	Normal mice	Manganese-treated mice	Surviving mice						
			Normal mice	Manganese-treated mice	Normal mice	Manganese-treated mice	Normal mice	Normal mice from manganese room	Manganese-treated mice
0	10	10	10	10	10	10	10	12	15
1	10	9	10	10	10	8	10	12	15
2	6	5	10	10	5	1	10	12	15
3	5	4	10	9	3	1	10	12	15
4	5	4	10	8	2	1	10	12	14
5	5	4	10	8	2	1	10	12	14
6	5	4	10	8	2	1	10	12	13
7	5	4	10	8	2	1	10	12	13
8	5	4	10	8	2	1	10	12	13

The results prove that the mice showed no difference in susceptibility to pneumococcus type II and IV after exposure to manganese, and the small difference in the mice injected with *Streptococcus Haem.* (Richards) may be due to the poorer condition of the mice exposed to manganese.

Absorption of Manganese.—Three mice exposed to manganese dust and 3 control mice were killed on the last day of exposure. All mice were carefully brushed, skinned and dissected.

Manganese content organisms for 3 mice	Manganese exposed		Control	
	3 mice	Average per mouse	3 mice	Average per mouse
Skins (including fur)	μgm. 74	μgm. 24·7	μgm. 30·6	μgm. 10·2
Brains ..	1	·3	1	·3
Livers ..	1	·3	1	·3
Lungs ..	1·2	·4	0·6	·2
Remainder of body excluding guts	lost in analysis			

IV. Manganese Dust (from Mill Room)

One hundred and twenty-four mice were divided into two groups: 63 placed in two cages for exposure to manganese dust, and 61 retained as controls. The dust was the same as used in Experiment III.

Weight of mice	Range gm.	Average gm.
Manganese exposed mice weighed	25–30	27
Control mice weighed	25–30	28

Thirty-three exposures to manganese dust were given: 17* exposed mice and 7 control mice died.

Bacteriological Results.—An attempt was made to set up pneumococcal infection of the lungs of the mice by spraying the air in enclosed chamber in which the mice were placed with a broth culture of pneumococci. No extensive experiments were, however, undertaken.

Culture.—A 24-hour serum broth culture of pneumococcus (type IV) was used. This culture was found to be virulent to mice only when given intraperitoneally in large doses.

Technique.†—The groups of mice were placed in small metal cages with open tops in the chamber, and broth containing pneumococci sprayed in through a hole at one side of the chamber, using a De Vilbis atomizer for this purpose. The spraying continued for 1 minute, then the mice were left exposed in the chamber for a further 15 minutes. At the end of this time they were removed and transferred to fresh cages.

Blood agar plates were exposed for 1 minute each, at intervals of 1, 5, 10 and 15 minutes after the spraying had ceased.

The results obtained are indicated below:

COUNTS OF ORGANISMS ON BLOOD AGAR PLATES IN CHAMBER AFTER SPRAYING WITH PNEUMOCOCCUS TYPE IV

1 minute after spraying. Plate absolutely covered with colonies.

* Shortly after this experiment began, intensely hot weather and room temperatures over 80° F. were experienced. This probably accounts for the high mortality in the exposed and control mice, but does not invalidate the purpose of this experiment to determine susceptibility to pneumococci.

† A similar technique had previously been used successfully for the investigation of aerosols.

Day	Daily length of exposure (starting at 9.30 a.m. and 2.30 p.m.) minutes	Dust count: particles per c.c.	Remarks
1	2×70	2216	
2	2×70	1555	
3	2×70	—	1 dead. No. P.M.
4	1×70	2381	1 dead. No P.M.
5	2×70	—	1 dead.
6	2×70	—	1 dead. *P.M.* Lungs normal. *Lungs section:* small subpleural peribronchia and periarterial areas of monocytic infiltration.
			Range gm. / Average gm.
			Manganese-exposed mice weighed .. .. .. 18–31 / 25
			Control mice weighed .. .. 23–32 / 28
7	2×70	2404	
8	2×70	—	
9 and 10	—	—	3 dead.
11	2×70	2637	
12	2×70	—	
13	2×70	1339	2 controls dead. *P.M.* Lungs normal. *Lungs section:* some polymorph and lymphocytic interstitial infiltration, few mononuclear cells; lung structure preserved.
14	2×70	—	1 dead. 1 control mouse dead. *Lungs section:* slight lymphocytic infiltration around bronchi. Remainder of lung normal.
15	2×70	—	2 dead.
16 and 17	—	—	1 dead.
18	2×70	2051	1 control mouse dead. *Lungs section:* areas of consolidation mostly due to lymphocytes and polymorphs; few mononuclear cells, ? pneumonia process due to temperature. Some dust cells.
19	2×70	1828	3 dead. (1) Eaten. (2) *P.M.* Nothing abnormal. *Lungs section:* subpleural areas of mononuclear infiltration; few dust cells. (3) *P.M. Lungs:* haemorrhagic areas. *Lungs section:* intense mononuclear proliferation especially around arteries, extending throughout lung.
20	2×70	—	1 dead. *P.M.* White patches on lung and liver. *Lungs section:* widespread and intense mononuclear infiltration with haemorrhage.
			Range gm. / Average gm.
			Manganese-exposed mice weighed .. .. .. 17–32 / 26
			Control mice weighed .. .. 17–35 / 25
21	2×70	2509	2 control mice dead. (1) Eaten. (2) *P.M.* Nothing abnormal. *Lungs section:* some polymorph infiltration; few dust cells.
	Exposures ended. Total exposures 33×70 minutes. 5 manganese mice (1 mouse showed numerous areas of consolidation in lungs) and 3 control mice killed for chemical analysis.		
22	—	—	1 ill—killed. *P.M.* Lungs apparently normal. *Lungs section:* peribronchial mononuclear infiltration; few polymorphs.

5 minutes after spraying.	Very large number of colonies.
10 minutes after spraying.	Large number of colonies.
15 minutes after spraying.	About 2000 colonies.

At the end of 15 minutes there were still pneumococci present in the air.

Control mice were treated in exactly the same way but were sprayed with sterile serum broth instead of pneumococci. They were exposed in the same chamber, being done before the pneumococcal mice. A blood-agar plate exposed for the full 15 minutes the mice were in the chamber gave a count of 50 organisms, all of which appeared to be of the usual air contamination type.

Experiment.—(1) Ten manganese-treated mice were exposed to pneumococcal infection for a period of 15 minutes.

(2) Ten normal mice were similarly treated.

(3) Ten manganese-treated mice were exposed to pneumococcal infection for 15 minutes on three different days, with an interval of a day in between each exposure.

(4) Ten normal mice were similarly treated.

Controls.—(5) Ten manganese-treated mice were sprayed with serum broth only and exposed for 15 minutes.

(6) Ten normal mice were similarly treated.

(7) Ten manganese mice were untreated.

(8) Ten normal mice were untreated.

TABLE 11

SURVIVING MICE—AFTER EXPOSURE TO PNEUMOCOCCAL INHALATION

Time after exposure	Pneumococci+broth 1 exposure		Pneumococci+broth 3 exposures		Broth only 1 exposure		Unexposed	
	Manganese mice	Normal mice	Manganese mice	Normal mice	Manganese mice	Normal mice	Manganese mice	Normal mice
0 day	10	10	10	10	10	10	10	10
1 ,,	10	10	10	10	9	10	10	10
2 days	10	10	10	10	9	10	10	10
3 ,,	10	10	10	10	9	10	10	10
4 ,,	10	9	10	10	9	10	10	9
7 ,,	10	9	10	10	9	10	10	9
2 weeks	10	9	10	10	9	9	see Table 12	
3 ,,	9	9	9	10	9	9		

The normal mice had been kept in the same room as the manganese chamber. The results are summarized in Table 11.

As it appeared after 7 days that under these relatively simple experimental conditions the manganese-exposed mice were not going to prove more susceptible to the pneumococcus infection, the two groups of unexposed control mice were removed and injected intraperitoneally with pneumococcus (type IV). Each mouse received 0·5 c.c. of suspension of organisms containing 220,000 organisms per ml. (Table 12).

TABLE 12

SURVIVING MICE INFECTED WITH PNEUMOCOCCUS (TYPE IV)

Time after infection	Manganese mice	Normal mice
0 day	10	9
1 ,,	10	9
2 days	9	8
3 ,,	9	8
4 ,,	8	6
6 ,,	7	6
7 ,,	6	6
2 weeks	6	6

These results also show no increase in susceptibility to pneumococcal infection in manganese-treated mice.

Absorption of Manganese.—Five exposed and 3 control mice were washed, skinned and dissected, and submitted for analysis.

	Manganese-exposed		Control	
	Average	Range	Average	Range
	μgms.	μgms.	μgms.	μgms.
Body ..	46	28–84	24	21–28
Skin ..	9·7	6·1–13·8	3·2	1·7–2·8
Brain ..	0·21	0·17–0·25	0·12	0·10–0·14
Lungs ..	0·2	0·15–0·28	0·03	0·01–0·05

Discussion

The incidence of pneumonia, observed in the present investigation, of 26 (range 15–63) per thousand in men exposed to the inhalation of manganese dioxide corresponds closely to that given by Buttner (1939) except that no fatal cases have been experienced. It seems very probable that the conditions in the factory described in this report were much better than in German mines or pyrolusite mills. No overt case of manganese poisoning has been observed (even after careful clinical examination of men exposed for 9 years), whereas reports of manganese intoxication are constantly appearing in the German literature.

No definite evidence of fibrotic or other specific changes have been found in the present investigation, and Schwartz (1942) agrees that manganese does not cause fibrosis. The fibrotic changes observed by Buttner (1939) are almost certainly due to 20 per cent. silica present in the Giessen pyrolusite.

Basic slag contains about 10 per cent. of manganese dioxide, and there have been many Continental reports of Thomasslachen pnuemonia. Gundel and Fischer (1938) state that 2 per cent. of basic slag workers die of pneumonia, and a sudden death in an agricultural worker (Koch, 1942) who had been employed in spreading basic slag has been ascribed to broncho-pneumonia caused by slag. As Jötten and Reploh (1938) remark, the factors in basic slag giving rise to pneumonia have to be elucidated. It would be wrong, at the present time, to necessarily incriminate manganese. Respiratory disease, including Thomasslachen pneumonia, in workers grinding basic slag and manufacturing artificial fertilizers (but not agricultural workers) has been a scheduled disease, for which compensation is payable, in Germany since 1936.

The dust present in the atmosphere studied in the present report, collected by an electrostatic precipitator, had a manganese dioxide content varying from 41 to 66 per cent. (The dust used for animal experiments was analogous, having a manganese dioxide content of 70 per cent.) The environmental studies, the first of their type to be reported,

are significant in showing the degree of exposure, and though the dust counts vary considerably and exposure is discontinuous high counts are not infrequent. Practically all particles were below 1μ in size, and roughly 80 per cent. below 0·2μ.

In the first animal experiment of the present report the dust had a high alkalinity and though this may have further increased the damage to the lung (or even laid open the way for the action of manganese), similar changes were induced on subsequent experiments with dust of low alkalinity. In practice, however, the alkalinity of the dust on the human lung cannot be discounted without further investigation (especially in view of the effect of basic slag).

The animal experiments were complicated by the very obvious toxic effect of the manganese; all animals exposed to manganese dust were, at the end of the experiment, in a less well condition than the control animals, and some exposed animals were in a frankly poor condition. Loss of weight occurred and the maximum exposure, which it appeared mice were able to tolerate, was 70 minutes twice daily for about 30 exposures at a dust count of about 2000 particles per c.c. of air. Even in their poor condition the exposed animals, under the conditions of the experiment, did not show any increased susceptibility to a pneumococcus type II or type IV administered by intraperitoneal injection and pneumococcus type IV by inhalation. No great increase in susceptibility to haemolytic streptococci was apparent.

The present experiments were undertaken with the object of determining whether manganese causes an increase in susceptibility to the pneumococcus. For the investigation of the effect of manganese on the lungs a larger animal would have been chosen, but the histological effects were so uniform and so striking that a detailed description is warranted.

The changes in the lungs were dependent on the length of exposure, and varied from slight mononuclear infiltration (an exaggeration of the normal) to intense mononuclear interstitial infiltration with many dust-laden cells, and finally to consolidation (with haemorrhagic areas), leading to complete disorganization of the lung structure. The changes appeared to occur especially around the bronchi.

The alveolar cells and the cells lining the bronchi were swollen and had undergone a hydropic change. The alveoli and interstitial spaces were packed with large mononuclear cells which were either desquamated alveolar cells or histocytes which had undergone intense proliferation. In many areas this monocytic reaction was so intense that only a faint outline of the normal lung structure could be seen. The mononuclear cells in some cases contained particles of dust and cell debris and appeared to be dead. Areas of necrosis were not uncommonly present in the lung. Lymphocytes were few in number and granular cells were conspicuous by their rarity (see fig. 10).

The oedema of the bronchial and alveolar epithelium suggests that manganese has a direct action on these cells. The presence of dust cells, though few in number, suggests that some particles of dust were phagocytosed in the normal manner but after absorption killed the phagocytic cells. Though dust-laden cells were observed in the bronchi, alveoli and interstitial tissues, none were observed in the lymphatic glands; this may be explained by the absorption or solution of manganese by cellular fluids.* (This agrees with Jötten's (1939) opinion that the action was local and that deep-seated interstitial changes do not occur.) Either before or after absorption manganese would appear to have a specific chemical action, causing intense mononuclear proliferation and infiltration, sufficient in many areas to cause consolidation of the lung and, as the action of manganese proceeds, cellular death, necrosis and haemorrhage.

From these facts it seems that following the inhalation of particles of manganese dust, phagocytosis proceeds in the usual way, but is overshadowed by the chemical action of manganese, and that this action results in a characteristic mononuclear celled proliferation.

No permanent pulmonary changes were observed in the men working in the factory, either on clinical or radiographic examination. Though naso-pharyngeal catarrh was common, attacks of bronchitis were frequent and in some men a spasmodic element gave rise to attacks similar to asthma, all changes eventually subsided after removal from exposure. It seems probable that in man the primary change is an oedematous reaction affecting the epithelium of any part of the respiratory tract. Further investigation is required to establish whether illnesses in the human subject diagnosed as pneumonia are due entirely to the chemical action of manganese or whether the irritative action of manganese allows

* To test this theory the solubility of manganese in serum was determined by Mr. N. H. Law, M.Sc., F.R.I.C.

Method. Manganese dust was sterilized by heating (T = 160° C.) for two hours. 0·4 gm. of dust was added, under sterile conditions, to 20 ml. of serum and mechanically shaken.

The suspended dust was separated by high-speed centrifuging and the supernatant liquid syphoned through a No. 32 Whatman filter paper. A 10 ml. aliquot was subjected to a pre-treatment of evaporation to dryness, ashing at a temperature not exceeding 600° C. sulphation and nitration, to ensure complete freedom from chloride, prior to the development of the permanganate colour using the method of High (1944). The colour was estimated photo-metrically by a Spekker absorptiometer using Ilford Spectrum Green Filters 604, a heat-absorbing Filter H. 503 and 4 cm. cells.

	Main Hall Dust	Mill Dust
	per cent.	per cent.
Alkalinity (as K_2CO_3)	12·4	0·03
Particle Size (less than 1μ diameter)	70	87
Solubility (mgm. per 100 ml.)—		
24 hours	0·34	1·18
48 hours	0·39	1·42
96 hours	0·27	1·37
144 hours	0·43	2·30

Control. Modified Ringer solution (King 1945) under exactly comparable conditions for the same times did not dissolve any manganese.

Accuracy. Results are accurate to ± 0·04 mgm. per 100 ml.

Conclusion. Manganese dioxide dust examined under the above conditions appears to dissolve in the serum but the dissolution is hindered by the presence of alkali. The fall in the amount dissolved at 96 hours followed by a steep rise at 144 hours suggests some change taking place in the serum.

secondary infection by the flora of the nose and mouth. Clinical experience suggests, and the histological appearance in mice confirms the suggestion that manganese has a specific action on the whole respiratory tract from the nose to the alveoli—the symptomatology depends on the part most affected and this in the present state of knowledge seems largely fortuitous. The term ' manganese pneumonia ' would seem to attach too much importance to anatomical considerations. Manganese pneumonitis is a better term.

The chemical observations were complicated by the small amount of manganese to be estimated, and the tediousness of the wet oxidation of the samples. No conclusion can be drawn other than that the mice exposed to manganese dust were absorbing manganese.

Summary

(1) Men exposed to the inhalation of manganese dust suffer a high pneumonia rate (averaging over the years 1938–45 inclusive 26 per thousand compared with 0·73 per thousand in a control group).

(2) For the purpose of exposing mice to the inhalation of manganese dust, a new dust-feed apparatus is described. After the inhalation of manganese dust, mice do not show an increased susceptibility to pneumococci or streptococci.

(3) After the inhalation of manganese dioxide dust, some particles may be phagocytosed in the normal manner, but all particles, whether phagocytosed or not, pass into solution in tissue fluids. Manganese is toxic to the respiratory epithelium and exerts a specific chemical action, which in mice results in an intense mononuclear proliferation and infiltration of the lung tissues and, as the action of manganese proceeds, in cellular death and necrosis.

(4) Manganese dust may affect any part of the respiratory tract, from the nose to the alveoli. Further investigations are needed to confirm the theory, suggested by these experiments, that manganese dust has a specific action on the human lung. Manganese pneumonitis appears a more appropriate term than manganese pneumonia.

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THE DISAPPEARANCE OF SULPHEMOGLOBIN FROM THE BLOOD OF TNT WORKERS IN RELATION TO THE DYNAMICS OF RED CELL DESTRUCTION

BY

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Preliminary studies of the rates of disappearance of Methemoglobin (MHb) and Sulphemoglobin (SHb) from human blood *in vivo* after cessation of contact with the agents, such as sulphonamides, acetanilide or TNT, causing these abnormal pigments (Jope and O'Brien (1942): Jope, O'Brien and Watson (1942)), revealed a striking contrast between MHb and SHb in this respect. MHb was found to disappear rapidly from the circulating blood and was undetectable after 2 to 5 days from removal of the causative agent (*cf.* also Cox and Wendel (1942)), whereas SHb was found to persist and was still detectable after many weeks. Methemoglobin also disappeared rapidly from drawn blood, being usually no longer detectable after about 24 hours at room temperature (Jope and O'Brien (1942)). Hence the persistence of MHb in the body during administration of the causative agent must be due to the interaction of two competing processes, the formation of MHb by interaction probably with some metabolite of the sulphonamide or TNT, and the reversion of MHb back to functional hemoglobin, a reduction of the ferric iron atom, which several workers have shown to be coupled to the intracellular reduction processes such as glycolysis, and to be inhibited by fluoride or iodoacetate ions (Warburg *et al.* (1930, 1931): Wendel (1933): Cox and Wendel (1942): and Drabkin (1945)). SHb, on the other hand, may be kept unchanged in drawn blood even longer than the red cells can be kept intact, and remains for several weeks after hemolysis as a constant fraction of the total pigment present, so long in fact as the blood can be kept sterile. It appears, therefore, that the red cell has no mechanism within it for the transformation of SHb. If this is so, the body can have no means of removing SHb other than by destruction of the red cells that contain it.

The disappearance of sulphemoglobin *in vivo* was therefore studied in some detail, both in order to gain a more precise knowledge of the conditions necessary for recovery from SHb cyanoses, and as a possible means of studying the dynamics of red cell destruction.

Material and Methods

Material. Seven TNT workers whose blood contained more than 4 per cent. of total pigment as SHb were selected for the experiment. Of those, 3 males and 2 females with an age range of 30 to 50 appeared to have no reaction to TNT contact other than cyanosis and could be regarded otherwise as normal healthy individuals. The other 2 males exhibited a more marked reaction to TNT after only a few weeks of contact. One was a man of 40 with a history of malaria and dysentry. His liver was enlarged, but had been so for many years. The other was a man of 60 who in 4 weeks' contact developed nausea and vomiting, dyspnoea and cyanosis (△ in fig. 2). During this period he developed an enlarged liver. His hemoglobin was low, being 70 per cent. (Haldane) and fell to 68 per cent. during his first four weeks off contact, but subsequently rose reaching 80 per cent. by the end of the 3½ months of the experiment. 100 per cent. Haldane as used here is defined in Macfarlane, O'Brien *et al.* (1944).

These workers were removed from contact with TNT and put on to light work for the duration of the experiment. Blood was collected by venepuncture using potassium oxalate as anticoagulant. Some initial semi-quantative examinations for abnormal pigments were made at the factory but the stability of SHb made this unnecessary during the experiment, and later samples were sent by passenger train or express post, being examined always within 24 hours of venepuncture, and often much sooner. This rapidity of transport is of course much more important when MHb is being studied, but the use of blood sugar bottles containing fluoride to inhibit glycolysis should be helpful in these cases.

Estimation of Sulphemoglobin

This has been carried out from the spectral absorption curves of dilutions of the bloods in 0·1 per cent. sodium chloride, which were thoroughly cleared by centrifugation. (In determining MHb a buffer must be used as the absorption curve is very sensitive to small variations in pH (e.g. Austin and Drabkin (1935): Jope (1941)).

Preparation of Blood Dilutions for Spectrophotometry

It is necessary to take the greatest care in obtaining perfectly transparent blood dilutions for spectrophotometry if SHb and MHb are to be estimated with any accuracy. This is particularly important when estimating small amounts, such as those encountered towards the end of the experiment, when results from slightly turbid solutions would have been of little value. From dilutions of

abnormal bloods it is often particularly difficult to remove the turbidity completely and Horecker (1944) has shown that the formation of MHb in the blood of men and cats by the action of TNT or related substances may be accompanied by the accumulation of denatured globin within the red cells, giving persistent turbidity on dilution. Although much turbidity can be avoided by using an alkaline diluent such as 0.1 per cent. sodium carbonate, in the present work it has been desirable to work in more neutral solutions as some abnormal blood pigments are less stable and their spectral absorption less marked in alkaline solutions. Accordingly, vigorous centrifugation of 0.1 per cent. sodium chloride dilutions has been used. Ten minutes spinning at 20,000 r.p.m. using an International high-speed head, which had previously been chilled in acetone-CO_2 has been found most satisfactory, though most dilutions can be cleared by spinning in an angle centrifuge for 20 minutes or less at 9000 r.p.m. At 6000 r.p.m. 40 to 60 minutes is often necessary, when heating effects become troublesome. The 0.1 per cent. sodium chloride diluent contained 0.5 per cent. sodium oleyl sulphate (MMB) to ensure complete hemolysis.

Spectrophotometry

The spectral absorption curves were obtained with a Hilger medium quartz spectrograph E498, and spekker photometer, using a 108-watt Tungsten ribbon filament lamp as a light source. Ilford panchromatic plates (H & D 500) were used, having an upper wavelength limit of sensitivity about $\lambda = 660$ mμ. Kodak IIIF emulsion is sensitive up to 690 mμ. A photo-electric microphotometer was used to observe the wavelength of the match points on the plates.

For the absorption curves in the red beyond $\lambda =$ 600 mμ 1/25, 1/10 or 1/5 dilutions of whole blood were examined in 5 cm., 1 cm., or 0·5 cm. cells respectively. For the range $\lambda = 450$–600 mμ, 1/25, dilutions were examined in 0·5 cm. cells. Under these conditions a blood containing 5 per cent. of its pigment as SHb gives extinction coefficients of about 1·3 at 540 mμ and 0·8 at 620 mμ.

The amounts of HbO_2 (x grams per cent.) and SHb (y grams per cent.) in the whole blood may be calculated from expressions (1) and (2).

grams per cent. HbO_2,

$$x = 0{\cdot}122\ E_{540} - 0{\cdot}0965\ E_{620} \quad . \quad (1)$$

grams per cent. SHb,

$$y = 0{\cdot}13\ E_{620} - 0{\cdot}00029\ E_{540} \quad . \quad (2)$$

where E_{540} and E_{620} are the extinction coefficients of the whole blood at $\lambda = 540$ and 620 mμ, i.e.

$$\frac{\text{observed optical density} \times \text{dilution of whole blood}}{\text{cell thickness in cms.}}$$

From these values of x and y the percentage of total pigment in the form of SHb may be calculated from the expression :

$$\text{SHb, percentage of total pigment} = \frac{y}{x+y} \times 100 \quad (3)$$

The constants in (1) and (2) must necessarily be fixed somewhat arbitrarily, as it is impossible to obtain a solution containing pure SHb uncontaminated with other absorbing pigments. For the particular purpose of this paper, however, uncertainty in the absolute amount of SHb present is not a serious limitation, for it is only necessary to follow the disappearance of the SHb band at $\lambda = 620$ mμ. It is possible from the constants used in (1) and (2) to give the clinician a reasonable estimate of the non-oxygen combining pigment present. Drabkin and Austin (1935) first attempted to define the spectral constants of SHb, but Lemberg, Holden, Legge and Lockwood (1942) have since shown that some choleglobin is formed simultaneously with SHb by the action of H_2S and oxygen on hemoglobin, and as Drabkin and Austin did not take this into consideration, their estimate of the extinction coefficient of SHb, E mol. ($\lambda = 620$ mμ) = 10,600, was probably too low.* Lemberg *et al.* (1942) studied the spectral absorption of SHbCO and from their data a value E mol. ($\lambda = 617$ mμ) = 16,000 may be calculated.

In the present work, where in some cases there were reasons for avoiding the CO-derivatives, a value for SHb, E mol. ($\lambda = 620$ mμ) = 13,000, was derived from the data of Lemberg *et al.* by determining the relation between the spectral absorption of bloods containing SHb and the same after conversion to HbCO + SHbCO, and this value has been used in the derivation of (1) and (2). Some confirmation of the spectrophotometric estimates of non-oxygen combining pigment present in two bloods containing SHb was obtained by comparison of iron analyses and oxygen combining powers (Jope, Jope and O'Brien, (1944)). The spectral absorption of SHb both in the visible and UV requires further study, as the results found in this preliminary study do not agree in detail with some reported in the literature (Drabkin and Austin (1935): Michel (1938): Lemberg *et al.* (1942)).

The Process of Formation and Nature of Sulphemoglobin

By the action of H_2S and O_2 on hemoglobin, a compound is formed with a spectral absorption band at $\lambda = 620$ mμ, the position of which is comparatively insensitive to changes in pH, and to many reagents such as KCN or ammonium sulphide, which affect the spectrum of MHb. The band is, however, shifted to $\lambda = 617$ mμ by the action of CO. The identification of this pigment with clinically encountered SHb rests upon the appearance in the spectrum of the latter of a similarly stable band at $\lambda = 620$ mμ, similarly shifted to $\lambda = 617$ mμ by treatment with CO. Both the artificial and natural pigments are unstable in 0.05N sodium hydroxide.

The process of SHb formation *in vivo* is not known, though it probably involves an oxidation and Michel (1938) has shown that at any rate *in vitro* the presence of soluble sulphides are necessary as a source of SH. The factors which favour SHb

*E mol. as used for heme pigments is defined as the molar extinction of a solution containing 1 gram atom of iron per litre, i.e. in the case of hemoglobin for a molecular unit of 17,000.

rather than MHb formation following ingestion of a large variety of amino- or nitro-benzene derivatives are quite unknown; SHb is most commonly observed clinically and in animals after acetanilide or phenacetin ingestion. With TNT or sulphonamides the number of cases in which SHb is found is about 2 per cent. of those in which MHb is observed.

The constitution of SHb is still uncertain, and many of the difficulties in elucidating it arise from the inability to produce the pigment in pure form (Lemberg *et al.* (1942)). That the general character of the protein in SHb is the same as that of the oxyhemoglobin from which it is formed is indicated by the electrophoretic homogeneity of the two pigments as shown by Michel (1938), and it has been found impossible so far to separate them by chromatography (Jope, Jope and O'Brien (1944)). Michel (1938) found it impossible to separate rat or dog SHb from oxyhemoglobin by crystallization, though Lemberg *et al.* (1942) have achieved such a separation in the case of SHb formed by sulphur and acetanilide ingestion in rats. The change in absorption spectrum indicates that the chief differences between SHb and HbO_2 may be expected in the heme group. It seems clear now that the prophyrin ring structure is unbroken in SHb (Lemberg *et al.* (1942): Jope (1941)). Haurowitz (1940) found that acid hydrolysis or peptic digestion of SHb did not give a separation of the heme group from the protein as it did with hemoglobin itself, but gave a compound with much protein material still attached to the heme, which he called Sulpheminproteose. From further drastic degradation experiments he suggested that some earlier findings of Haurowitz was due to the presence of free sulphir in the latter's product, which was eliminated cataphoretically by Michel.

SHb can combine quantitively with CO (Michel (1938): Jope, Jope and O'Brien (1944)) and the central iron atom appears to be in the ferrous state, which may also be concluded from Keilin's (1933) demonstration of the existence of Sulphmethemoglobin.

Results

Disappearance of SHb *in vivo*

The results from the study of the seven TNT workers are shown in fig. 2. To reduce all the data to a comparable vertical scale, the SHb has been plotted as the percentage of the amount present on the day the worker was removed from contact with TNT, the time, t, is in days subsequent. From all the data the regression of SHb on time has been calculated as

$$SHb = 96 - 0{\cdot}83t \quad . \quad . \quad . \quad . \quad (4).$$

The actual levels of SHb encountered in these workers during contact with TNT were between 4 and 7 per cent. of total blood pigment. The observation of Lemberg *et al.* (1942) that no cholegloblin was detectable in clinical sulphemoglobinemia was confirmed in the present work.

Disappearance of MHb *in vivo*

The disappearance of MHb from the blood of a TNT worker who developed cyanosis soon after contact with TNT is shown by the broken line in fig. B. 12 per cent. of his total pigment was in the form of MHb at the end of contact, and MHb was no longer detectable five days later.

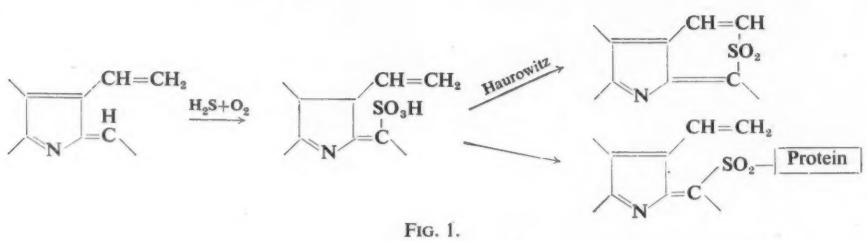

FIG. 1.

two $-SO_2H$ groups had become attached to two of the methene bridges of each porphyrin ring and that subsequent condensation had taken place with the vinyl groups. This does not account for the strengthened linkage between the heme and the protein. A possible explanation of this might, however, be found in a condensation with loss of water taking place between the $-SO_3H$ and a basic group of the protein (fig. 1).

Michel (1938) found one extra S atom per Fe atom in SHb but Haurowitz (1940) found two, and made the suggestion that a compound with the same spectral absorption would result whether one or two methene bridges of the porphyrin ring were substituted. This is improbable. Michel considered that the discrepancy between his results and

The Stability of Sulphemoglobin

In order to test the stability of intracellular SHb attempts were made to store bloods from some of these workers in an incubator at 37° C. Great difficulty was experienced in keeping the bloods sterile for more than a few hours under these conditions, as the addition of any sterilizing agent might have inhibited any intracellular enzyme which could transform SHb. Some success was, however, achieved in two experiments, though even here much MHb had been formed and over 80 per cent. of the red cells had been hemolysed by the end of ten days. The presence of MHb made it necessary here to reduce with sodium hydrosulphite and estimate the SHb using CO derivatives (Lemberg *et al.* (1942).) The observed SHb levels in these two

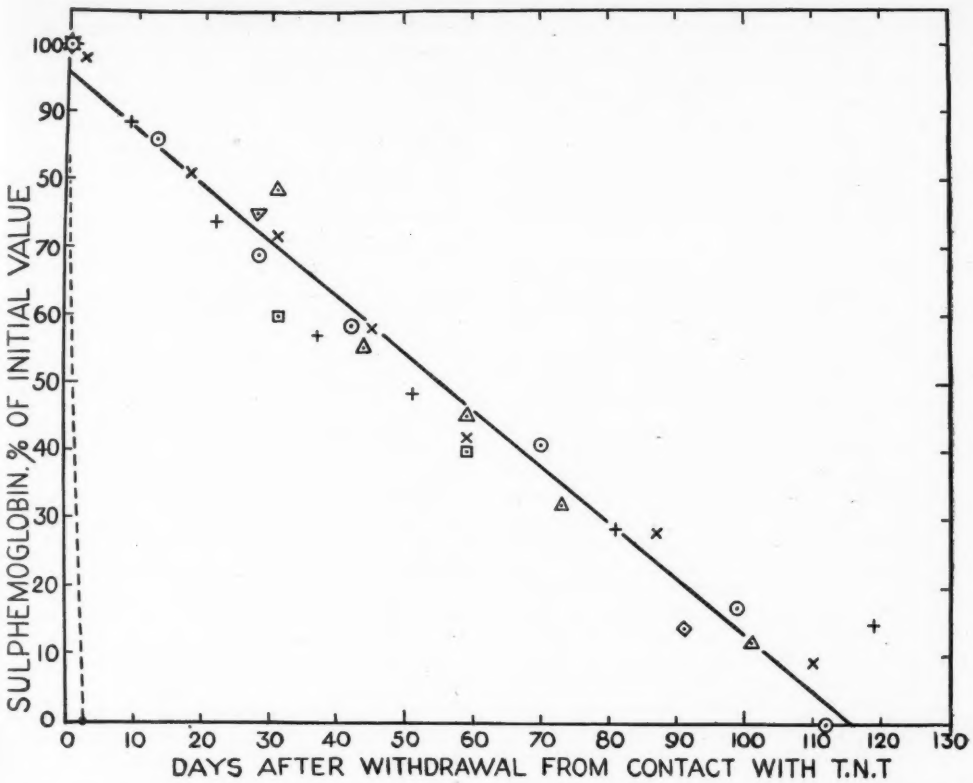

FIG. 2.—Disappearance of Sulphemoglobin and Methemoglobin from the blood of TNT workers after withdrawal from contact. The different representations of points along the Sulphemoglobin curve correspond to the seven different workers studied.

———————— Sulphemoglobin.
— — — — — — Methemoglobin.

cases remained as constant as could be expected under such conditions (Table 1). With further experience, and perhaps the addition of glucose to the red cell suspensions it might be possible to improve the quality of results from this type of experiment.

TABLE 1

THE STABILITY OF SULPHEMOGLOBIN AT 37° C. *in vitro*.

Days		0	2	5	10
SHb (per cent. of total pigment)	I.	7·2	7·2	6·9	7·5
	II.	5·0	5·0	—	5·8

Discussion

Life Span of the Red Cell

The greatest significance of these results lies in the connection between the disappearance of SHb and the destruction of the red cells which contain it. In relating these the following assumptions must be made :

(1) That the intact red cell possesses no means of changing SHb, and that the body has no means of removing it other than removal of the red cells which contain it.

(2) That no significant SHb formation goes on after administration of the apparent causative agent is stopped.

(3) That neither SHb nor the heme part of its molecule are incorporated into new red cells.

In relating the destruction of the red cells containing SHb with that of normal red cells, two further assumptions must be made :—

(1) That the SHb formed is initially distributed at random among the red cells of all ages existing in the body at that time.

(2) That the formation of SHb within the cell does not prolong or curtail its life.

It is difficult to verify assumption (1) directly. The experiments on storing blood containing SHb formed *in vivo* reported above, as far as they go show that naturally formed SHb is stable under sterile conditions both in red cells and in hemolysed solutions, but working at body temperature it is not possible to make such experiments last much more than a week. Moreover, red cells can hardly

be considered as existing reasonably unchanged for more than a day or so when whole blood is stored at body temperature. At lower temperatures the result would be hardly applicable here. The best test of assumption (1) probably results from a comparison of the forms of curves given by the differential agglutination and the marked nitrogen techniques (see below p.140), with those found for the disappearance of SHb in such conditions as hemolytic anaemias, etc. (e.g. Brown, Hayward, Powell and Witts (1944)).

Assumption (2) requires detailed study (*cf.* Cox and Wendel (1942) for MHb). By plotting in detail the SHb levels in animal experiments just before and after time = 0 (fig. 2), more information should be obtainable concerning the formation of SHb by various compounds, and the extent to which the point $t = 0$ is made uncertain due to continued formation of SHb by the toxic agent or a metabolite remaining in the body after administration is stopped might be assessed. However, this uncertainty of zero probably does not extend over more than a few days, and is perhaps comparable to the uncertainty of zero of the differential agglutination experiments on normal human subjects so far reported by Callender, Powell and Witts (1945), where they used donor's blood anything from 0 to 6 days old. The rapid disappearance of MHb from the human system on cessation of TNT (fig. B), may be significant in this connection, though it cannot be assumed that SHb and MHb are formed by the action of the same metabolic products of TNT and other toxic agents, and TNT or its metabolic products may be stored in the tissues for longer periods after prolonged contact..

Recent evidence shows that both the protein (Robscheit-Robbins *et al.* (1943): Whipple (1944)), and the heme (Bloch and Rittenberg (1945): Shemin and Rittenberg (1945)) of hemoglobin are formed from simple precursors during the maturation of the red cell, and there is no evidence to suggest that assumption (3) should be regarded as untrue.

Assumption (4) is also unsusceptible of simple proof. A single red cell containing 5 per cent. of its hemoglobin in the form of SHb would exhibit a SHb spectral absorption band at 620 mμ of optical density of the order 0·005, or an absorption of about 1 per cent. of the incident light, which could be measured by the methods devised by Caspersson (1936: 1940), but following the distribution of SHb between red cells in this manner over a long period would be a laborious process.

Assumption (5) must be regarded as reasonable as the formation of abnormal pigment within a red cell might be expected to curtail rather than lengthen its life, and in fact the life span derived from the present study is considerably longer than that derived from most earlier methods which were until recently generally accepted.

The observations of SHb level against time derived from these seven TNT workers are well fitted by the straight line $SHb = 96 - 0.83t$. If the above assumptions are correct, it will be seen from the mathematical treatment of red cell survival set out by Callender, Powell and Witts (1945) which is exactly applicable here, that the value of t at SHb = 0 is 116 ($\pm$ 5) days, and this represents the average life span of the red cell as indicated by the SHb method. These conclusions as to the life of the red cell and the linearity of decay are in close agreement with those of Callender, Powell and Witts (1945), who found an average total life span of about 120 days. Recently also Shemin and Rittenberg (1945) have studied the incorporation of the N^{15} isotope from ingested glycine into the protoporphyrin of hemoglobin, and have concluded that the life span of the red cell must be greater than 100 days. Their data appear to be consistent with the linear decay curves described above. It is greatly to be hoped that these workers will repeat this experiment and continue it long enough (160 days) to provide a further test of the conclusion that the life span of the red cell is about 120 days.

Variations in rates of formation or destruction of red cells will affect the linearity of the observed decay curve, and may lead to a curvature or a saw-edged character according as there is a regular drift or an erratic variation. Such erratic variation may possibly be operating in the case of the man of 60 whose hemoglobin was low (p. 136 and △ in fig. 2), but is hardly sufficient to affect greatly the general conclusions.

The observed linear decay implies that red cells have a fixed life within small limits and are not subjected to random destruction, which would lead to a logarithmic curve of decay. There are, however, indications from each of the above three experimental approaches that a small amount of random destruction may go on even in normal individuals. Callender, Powell and Witts attributed the slight curvature of the early portions of two of their normal curves to disturbances following transfusion, which is probably correct. In the SHb results the true SHb disappearance curve must clearly cut the $t = 0$ axis at SHb = 100, whereas the calculated regression line cuts at SHb = 96, thus suggesting a slight curvature, though the results are unfortunately hardly sufficient to warrant more elaborate analysis. In Shemin and Rittenberg's experiment the curve for 'atom per cent. N^{15}' against time reaches a maximum after 33 days, and the linear hypothesis requires that it then shall have a flat top until 120 days, or whatever the life span of the red cell should be, and then descend rapidly, reaching zero once more after about 150 days. Their results indicate that the top has in fact a slight negative slope, which might be due to some random destruction of red cells. Shemin and Rittenberg appear to be satisfied that no interchange of N^{15} takes place between the protoporphyrin and other molecules. These are all, however, only slight deviations from linearity, and in general the linear decay curve implying a fixed red cell life of about 120 days seems well established by these three experimental approaches.

The Differential Agglutination and Sulphemoglobin methods are complementary: in the former destruction of normal red cells can be followed, but not that of the subject's own cells, while in the latter the destruction of the subject's own red cells is followed, though having SHb formed within them they cannot be regarded as perfectly normal. Moreover, the Agglutination method is based upon a property of the cell envelope, and the Sulphemoglobin method upon a property of the cell contents. It is therefore particularly significant that they should lead to a similar result.

The three approaches discussed above, moreover, all give information concerning the dynamics of red cell destruction, whereas most earlier approaches to the problem of red cell life merely yield an estimate of the average life with no information as to the shape of the decay curve.

Most other methods which have been used or estimating red cell life have given much shorter times—30 to 50 days in man. These have been critically reviewed recently (Callender, Powell and Witts (1945): *Lancet* (1945): *British Medical Journal* (1946)), and only a few remarks need be added here. Assuming that it is possible to estimate the *in vivo* reticulocyte maturation time from observations in culture solutions (Barr and Lloyd (1943)), it would only be possible to derive an estimate of red cell life from this if the average value for the reticulocyte count were based upon a statistical study over a considerable time, which does not so far appear to have been attempted. Estimates of red cell life based on the estimation of excreted pyrrole pigments arising from the breakdown of hemoglobin are subject to uncertainty whether all the pyrrole portion of the heme of hemoglobin from destroyed red cells is transformed into a form which can be determined, to great difficulties of estimation, and also to the hitherto undefined contribution from the breakdown of the muscle hemoglobin, which forms a considerable portion of the total hemoglobin pigment of the body.

It would be most valuable to apply the SHb method to following the destruction of red blood cells in animals, where the blood sub-groups are not specific enough for the differential agglutination technique to be used. It appears that SHb can be produced with some certainty in the rabbit or rat by feeding with acetanilide or phenacetin (Michel (1938) : Lemberg *et al.* (1942)), but that choleglobin may be formed simultaneously in amounts which might be sufficient to confuse the interpretation of the data. This choleglobin formation is especially marked with large doses of acetanilide given over a short period with the intention of producing sudden high concentrations of SHb, and hence a sharp point $t = 0$ on the SHb disappearance curve. In human clinical sulphemoglobinemia only negligible amounts of choleglobin are normally encountered: it is possibly produced under milder conditions and removed nearly as fast as it is formed, for it seems that choleglobin can be transformed intracellularly. In animal experiments it would be necessary to use a precise spectrophotometric method for estimation of SHb in order to eliminate interference from choleglobin.

Recovery from Cyanosis

If cyanosis is due to MHb as much as 35 per cent. of the total blood pigment may be in a non-oxygen carrying form, but recovery after removal of the causative agent is effected rapidly by the reducing enzyme systems within the red cells. This may be complete in slight cases in 24 hours, and in more severe cases in 4 or 5 days. It has already been pointed out (Rimington (1943)) that MHb cyanosis should perhaps be regarded more seriously than the mere fraction of blood pigment immobilized would suggest, as Darling and Roughton (1942) have shown that MHb affects the dissociation curve for $HbO_2 \rightleftharpoons Hb + O_2$ in a manner similar to the effect of HbCO (Douglas, Haldane and Haldane (1912)), making the oxygen less available to the tissues.

In cyanoses due to SHb rarely more than 7 per cent of the blood pigment is immobilized, but the effect of SHb on the dissociation curve of the remaining HbO_2 has not so far been studied. Although the amounts of SHb encountered are slight, the present work shows that it is over three months before it is completely eliminated from the system : it would, therefore, be of value to have a more precise knowledge of any probable toxic effects it may exert. SHb is more effective in producing the impression of cyanosis than a similar amount of MHb, as its absorption in the red is greater than that of MHb.

It must be emphasized that in order to distinguish between cyanoses produced by MHb and SHb careful measurements must be made on a good spectroscope, for instance, a Hartridge reversion spectroscope. If the wavelength calibration is inadequate the stability of SHb (band at 620 mμ) to reagents such as KCN (carefully neutralized) and sodium hydrosulphite may be used to distinguish it from MHb, whose 635 mμ band disappears with these reagents. With good illumination in a Hartridge spectroscope using a 1/4 dilution of blood in a 2 cm. layer about 3 per cent. of MHb and about 1 per cent. of SHb can be detected by their bands at 635 mμ and 620 mμ.

In our experience with TNT we have encountered about one case showing SHb to 40 showing MHb (Jope and O'Brien (1942)).

After all the cyanoses which we have been able to account for by the presence of MHb and SHb, and the few subsequently discovered to be suffering from a lung or heart complaint, there still remain some in which no abnormal pigment can be demonstrated, thus confirming the finding of Horecker (1944) and others. This has sometimes been thought to be due to a pigment, possibly a combination of a metabolite of the toxic agent with hemoglobin (*cf.* Keilin and Hartree 1943)), which immediately splits up on dilution. Examination on the spectrograph of thin films of red cells in some

such cases has failed to show up any spectral absorption bands in the red, and cyanosis was therefore probably due in those cases to excessive amounts of reduced hemoglobin in the peripheral blood vessels. The toxic agent or metabolite might well itself affect the O_2 dissociation curve of HbO_2, and this point might profitably be investigated. However, a loose compound between a metabolite of the toxic agent and the Hb, with a spectral absorption band on the red may well occur in some other cases.

Summary

(1) Whereas methemoglobin is removed from the circulating blood by intracellular reducing enzymes in 2 to 5 days after a TNT worker is taken off contact, Sulphemoglobin persists for about 116 days.

(2) The disappearance of sulphemoglobin from the blood of seven TNT workers has been related to the destruction of the red blood cells which contain it, the necessary assumptions being discussed. A linear decay is found, disproving random destruction and indicating that the red cell has a fixed life-span. This is estimated at 116 days. These conclusions are in agreement with other recent work.

(3) Possible applications of this method of studying the dynamics of red cell destruction are discussed.

(4) Recovery from cyanosis is discussed.

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EFFECTS OF HEAT ON WIRELESS TELEGRAPHY OPERATORS HEARING AND RECORDING MORSE MESSAGES

BY

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When the Royal Navy asked the Medical Research Council to enquire into the effects of heat on human beings, the resulting investigations came under two general headings. The first was the question whether high atmospheric temperatures affected some forms of human activity more than others. An answer to this question was needed to determine which compartments in ships should have priority in the allocation of air-conditioning equipment. The second main aim of the research was to discover the extent of the overlap between the effects of the heat on the body as assessed by physiological techniques and the effects of heat on human performance as measured by psychological experiment. Answers to this second question might indicate how far it was safe to use simple measures of bodily functions to predict the presence of performance deterioration.

The first question has entailed studies of the effects of heat on a wide variety of real-life human activities, all of which are vital for the fighting efficiency of the modern warship. Actual human performance has been measured with tests demanding either hard thinking or heavy physical effort. Sometimes, however, a different line of approach has been tried with the volunteer experimental subjects who were usually Naval ratings; instead of these men having too much to do, their patience has been tried by giving them too little to do, as, for example, in jobs imitating dull and monotonous look-out duties. The accuracy of their watch-keeping showed whether they could remain wide awake and alert under these circumstances. Conversely, the ability to sleep soundly with the minimum of restlessness has also been studied at different room temperatures. But there is no great difference between the effects of heat on all these various forms of human activity, and this paper can therefore be taken as representative of the bulk of the evidence now available—see also Houghten (1942), and Weiner and Hutchinson (1945).

Specifically to this study, the problems relating to the first main question were:

1. Do hot and moist atmospheres impair the accuracy with which trained wireless telegraph operators can write down morse messages heard over their headphones?

2. If any such deterioration is found, how far may the atmospheric temperature be allowed to rise (above the lowest temperature readily obtainable with air-conditioning plant in tropical waters) before this deterioration becomes statistically definite?

The second general question of the correlation between bodily changes and human performance is clearly a complex matter and here the research aims have been limited to an analysis of the relationship between rectal temperature and working efficiency since rectal temperature is one of the most widely used physiological measures in hot-room investigations. The specific problems arising from this second main question were:

3. Does increased room temperature lead to a rise in body temperature with sedentary work of this nature?

4. Are the performance test scores on this wireless telegraphy reception test statistically related to the rectal temperature readings?

5. If so, is there any evidence that a rise in body temperature causes deterioration in ability on this wireless telegraphy test?

Main Findings

1. Hot and moist atmospheres seriously impaired the accuracy with which trained wireless operators could record morse messages heard over their headphones.

2. A statistically reliable reduction in wireless telegraphy reception accuracy first appeared when trained and acclimatized men were exposed to an atmospheric temperature of 87·5° F. on the effective temperature scale, i.e. *performance deterioration became statistically definite when the wet bulb reading was* 85° *F. and the dry bulb was* 95° *F. with an air velocity of* 100 *feet per minute.* In the First Report of the Habitability Mission to the Eastern Fleet, Surgeon Lieutenant-Commander F. P. Ellis, O.B.E., R.N., noted that the majority of the wireless telegraphy ratings in the Eastern Fleet had to work at effective temperatures which were higher than 84° F., and also that the conditions were known to be even more severe during prolonged spells at action stations (Ellis, 1946).

3. Hot and moist atmospheres raised the rectal

temperatures of men recording wireless telegraphy messages.

4. Though the rectal temperature was remarkably sensitive to the effects of heat and high humidity, it did not give even an approximate idea of the effects of hot and humid atmospheres on the accuracy of work at W.T. reception.

5. Raised body temperature did not therefore cause deterioration in ability at W.T. reception, within the range of the atmospheric conditions encountered by ratings in the Eastern Fleet.

Procedure

Experienced Subjects

The general experimental arrangements are as described in Appendix III. The experimental subjects were eleven physically fit volunteers whose ages ranged between 18 years and 34 years as in Appendix I. Their previous experience varied considerably: eight of the operators were ex-overseas R.A.F. personnel, all of whom had just returned from 3 years' W.T. operating abroad—Subject No. 1 had been in Ceylon; No. 2 in Aden; Nos. 3, 4, 5, 7 and 10 in the Middle East; and No. 11 in the Middle East and Persia. Subject No. 6 had done 4 years' W.T. operating in this country and Nos. 8 and 9 had had only 1 year's W.T. experience in this country and had not been abroad.

Further W.T. Practice

On arrival at the laboratory further extensive practice in W.T. reception was given. The subjects were tested in two separate groups—Group A (Nos. 1, 2, 4, 5 and 6) and Group B (Nos. 3, 7, 8, 9, 10 and 11). Group A had 5 weeks' practice consisting of 3 hours W.T. reception a day for 6 days a week. Group B had 7 weeks of similar practice.

Acclimatization

Group A had previously spent 6 weeks in the hot room as they took part in other experiments. The majority of the exposures were between 87·5° F. and 97° F. for about three hours a day, 5 days a week. In addition to this, Group A had a further 5 weeks in the heat before being tested, since all their W.T. practice spells were in the hot room. Altogether therefore Group A had had 11 weeks preliminary acclimatization before the main W.T. test was given, and in most of the exposures they experienced effective temperatures of between 87·5° F. and 97° F. for about three hours a day. Bedford (1944) has explained the use of the effective temperature scale in a previous paper.

Group B also did their preliminary W.T. reception practice in the hot room. This consisted of 3 hours a day, 6 days a week. They were therefore exposed to effective temperatures which were mostly between 87·5° F. and 97° F. during their preliminary acclimatization spell of 7 weeks before the main W.T. tests began.

Test Days

In the actual test, Group A worked at W.T. reception in five different atmospheric temperatures. On five different days the dry bulb/wet bulb readings were either 85°/75°, 90°/80°, 95°/85°, 100°/90° or 105°/95°, these temperatures being held to within ±1° F. on any particular day. As the wind velocity was 100 feet per minute the readings on the effective temperature scale were 79° F., 83° F., 87·5° F., 92° F. and 97° F. Group A experienced these five room temperatures once on five successive days. They had the lowest temperature on the first day and worked up to the highest temperature for the fifth day.

During their test Group B had the same five room temperatures, but each temperature was given twice on successive days, i.e. on the Monday and Tuesday the effective temperature was 79° F., on Wednesday and Thursday it was 83° F., on Friday and Saturday it was 87·5° F. On the following Tuesday and Wednesday it was raised to 92° F. and on Thursday and Friday it was 97° F.

The test period on any one of the test days lasted for 3 hours. Nine messages were sent in that time and as each of these messages consisted of 250 groups just over 16 minutes was needed to transmit each message at the speed of 22 words per minute. About three minutes elapsed between each of the messages.

Rectal Temperature

Rectal temperatures were taken at the beginning and end of each of the 3-hour tests, which were always given in the morning.

Experimental Details

The men wrote the morse messages heard on their headphones while seated at a table and dressed in gym shoes and tropical shorts made from a white drill material. A sweat rag was provided and plenty of unsalted water was available. They all had a daily ration of 10 gm. of salt in addition to their ordinary dietary intake.

Until the third and final check of their answer papers (which was often a week or two after the event) the men had no precise knowledge of their level of accuracy on a particular day.

Results

These have been considered under three main headings:

(1) W.T. Reception Test Scores.
(2) Rectal Temperature Readings.
(3) Relationship between W.T. Scores, Rectal Temperatures and Atmospheric Temperatures.

1. W.T. Reception Test Scores

Nature of Work. Each of the nine messages given on any 3-hour test day consisted of 250 groups of five character ' syko ' (random mixed letters and numbers). In other words, each message transcribed in 16 minutes consisted of 1250 symbols, two-thirds of which were letters and one-third

FIG. 1.—Test in progress inside hot room.

numbers. This must be one of the quickest forms of work that men are called on to do; W.T. reception in this form might well be regarded as a supreme example of high-speed work since each man dealt with 3750 letters or numbers per hour.

Accuracy Criterion. Any letters or figures missing or found to be wrong on the written answer sheets were regarded as mistakes, and the number of these errors was taken as an index of the proficiency of the individual because the work was done at a fixed speed. A further point was that in calculating the average levels of accuracy attained by all the eleven subjects taken together, it seemed desirable, since Group A subjects were tested only once at each room temperature, and Group B men were tested on two occasions at each room temperature, to average the scores obtained on the two days by Group B so that these men should not have an undue influence on the average readings of all the subjects.

Average Scores obtained from the Arithmetic Means. Taking all the subjects together the average number of errors made per subject per hour showed that there was a definite increase in the number of mistakes at the higher atmospheric temperatures.

A statistical test of the significance of difference between proportions showed that the slight difference between the average W.T. error score at 85°/75° (79° ET) and that at 90°/80° (83° ET) was due to chance variations in the experiment, the ratio of the difference to its standard error was 0·8. *When the room temperature was raised to* 95°/85° (87·5° *ET*) *there was a statistically definite fall in accuracy compared with performance in the room temperatures of either* 85°/75° (79° *ET*) or 90°/80° (83° *ET*), the critical ratios here being 5·7 and 4·4 respectively.

AVERAGE NUMBER OF MISTAKES PER HOUR PER SUBJECT—ARITHMETIC MEANS

Dry bulb/Wet bulb room temperature	85°/75° (79° ET)	90°/80° (83° ET)	95°/85° (87·5°ET	100°/90° (92° ET)	105°/95° (97° ET)
Average number of mistakes per subject per hour.	12·0 errors	11·5 errors	15·3 errors	17·3 errors	94·7 errors

The question then arose whether it was desirable to consider all the subjects together in view of the large differences in operating ability even within this group of men, most of whom were highly trained. The subjects were therefore arranged in rank of order of ability on the basis of their performance at the two lowest temperatures, 79° ET and 83° ET. The best three men were classed as being 'exceptionally skilled', the next five as 'very good' and the last three as 'competent', these being entirely arbitrary terms.

A study was then made of the average number of

mistakes per subject per hour for these three categories under the various conditions of heat and high humidity.

AVERAGE NUMBER OF MISTAKES PER SUBJECT PER HOUR—ARITHMETIC MEANS

Efficiency category	Room temperature				
	85°/75° (79° ET)	90°/80° (83° ET)	95°/85° (87·5° ET)	100°/90° (92° ET)	105°/95° (97° ET)
'Exceptionally skilled' group (Subjects 1–3).	2·9 errors	2·7 errors	2·5 errors	1·9 errors	6·2 errors
'Very good' group (Subjects 4–8)	6·2 errors	6·6 errors	7·9 errors	12·0 errors	99·8 errors
'Competent' group (Subjects 9–11).	30·9 errors	28·7 errors	40·7 errors	41·8 errors	174·9 errors

These data have also been given in fig. 2. If one then calculates the *increase* in average number of errors per subject per hour by subtracting the average error score made at 85°/75° from the number of mistakes at the higher temperatures the following table is obtained:

ADDITIONAL NUMBER OF MISTAKES PER SUBJECT PER HOUR WHEN PERFORMANCE AT 85°/75° IS TAKEN AS THE GROUP STANDARD

Efficiency category	Room temperature			
	90°/80° (83° ET)	95°/85° (87·5° ET)	100°/90° (92° ET)	105°/95° (97° ET)
'Exceptionally skilled' group (Subjects 1–3).	−0·2 errors	−0·4 errors	−1·0 errors	+3·3 errors
'Very good' group (Subjects 4–8).	+0·4 errors	+1·7 errors	+5·8 errors	+93·6 errors
'Competent' group (Subjects 9–11).	−2·2 errors	+9·8 errors	+10·9 errors	+144·0 errors

In other words, the level of operating efficiency attained by the subjects determines the extent of their work deterioration at a given atmospheric temperature.

Average Scores obtained from Median Readings

To ensure, therefore, that too much emphasis was not being placed on the performance deterioration of men at the less efficient end of the scale, it seemed necessary to estimate the average error scores by the median readings. This estimate of the average is obtained by arranging the group of scores concerned in order of merit, and then taking the central observation of this rank order (or the average of the two central readings).

AVERAGE NUMBER OF MISTAKES PER SUBJECT PER MESSAGE—MEDIAN READINGS

Efficiency category	Room temperature				
	85°/75° (79° ET)	90°/80° (83° ET)	95°/85° (87·5° ET)	100°/90° (92° ET)	105°/95° (97° ET)
'Exceptionally skilled' group.	0·5 errors	0·0 errors	0·0 errors	0·5 errors	1·0 errors
'Very good' group.	1·5 errors	1·5 errors	2·0 errors	2·0 errors	4·5 errors
'Competent' group.	5·0 errors	7·5 errors	8·0 errors	10·5 errors	9·0 errors

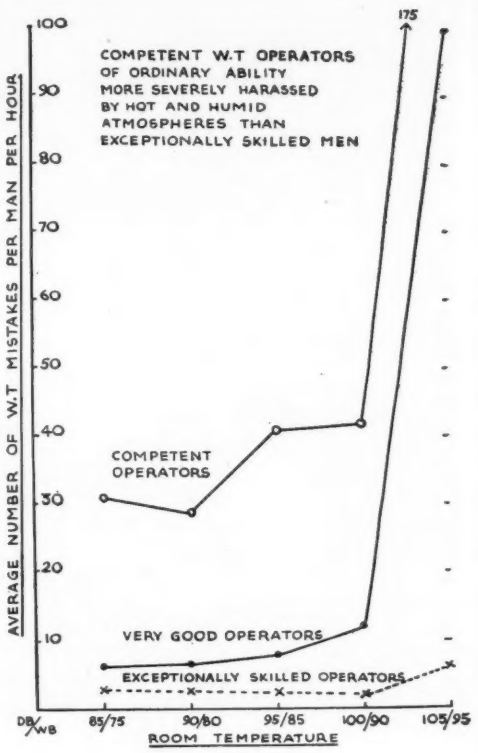

FIG. 2.

Since three messages were dealt with in an hour these readings were multiplied by three to give the average number of mistakes per subject *per hour*.

AVERAGE NUMBER OF MISTAKES PER SUBJECT PER HOUR—MEDIAN READINGS

Efficiency category	Room temperature				
	85°/75° (79° ET)	90°/80° (83° ET)	95°/85° (87·5° ET)	100°/90° (92° ET)	105°/95° (97° ET)
'Exceptionally skilled' group.	1·5 errors	0·0 errors	0·0 errors	1·5 errors	3·0 errors
'Very good' group.	4·5 errors	4·5 errors	6·0 errors	6·0 errors	13·5 errors
'Competent' group.	15·0 errors	22·5 errors	24·0 errors	31·5 errors	27·0 errors

Incidence of Faulty Messages

A third way of analysing the results was to count the number of written messages falling below a given standard at each of the five atmospheric temperatures. Altogether 99 of these messages were available for each room temperature since each of the eleven subjects provided nine of these messages per room temperature. The eighteen answer messages from each member of Group B were averaged to give nine representative scores per subject per room temperature.

Each message consisted of 250 five-symbol groups

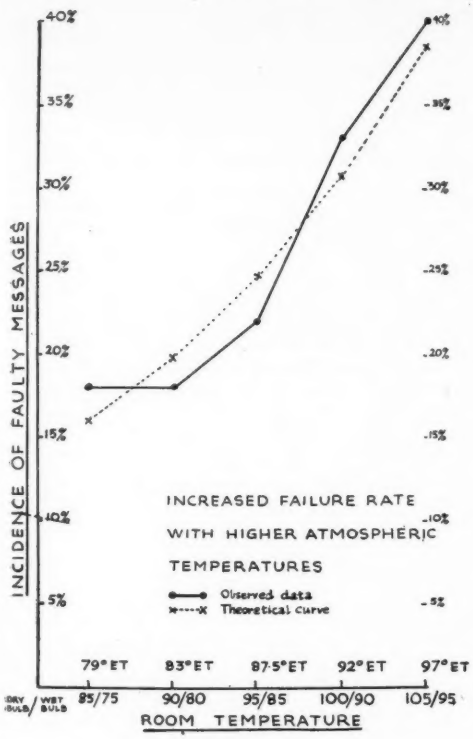

FIG. 3.

and any such message with five symbols wrong or missing (either letters or numbers) was regarded as faulty. As there were 99 messages available for each room temperature, this number very nearly represents the percentage incidence of faulty messages. These results are also shown in fig. 3.

Dry bulb/Wet bulb effective temp.	Room temperature				
	85°/75° (79° ET)	90°/80° (83° ET)	95°/85° (87·5° ET)	100°/90° (92° ET)	105°/95° (97° ET)
Incidence of faulty messages (more than five mistakes per message).	18% (18/99)	18% (18/99)	22% (22/99)	33% (33/99)	40% (40/99)

The observed data suggested that deterioration as measured by the incidence of faulty messages bore a logarithmic relation to atmospheric temperature changes. This was tested by fitting a curve of the general formula

$$\log Y = c_0 + c_1 x$$

where c_0 and c_1 were constants, x room temperature and Y the incidence of faulty messages. These constants were calculated from the observed data by the usual methods. The goodness of fit of the theoretically constructed curve to the observed data was examined by the analysis of variance and found to be highly satisfactory. The actual technique employed is described in Goulden's 'Methods of Statistical Analysis' on pp. 231–34. The equation for the theoretical curve shown in fig. 3 was

$$\log Y = 1{\cdot}10768 + 0{\cdot}09568x.$$

It was most interesting that previous research with the heavy pursuitmeter gave the same general formula for deterioration in the heat. With this entirely different form of work in which the task was to move a heavy lever with precision, the formula was $\log Y = c_0 + c_1 x$ when c_0 and c_1 were constants, x room temperature and Y the incidence of unsuccessful attempts.

With this theoretical curve it was possible to have a second estimate of the temperature at which W.T. reception was significantly worse proportionally than that obtained at 85°/75°, which was regarded as the lowest practicable temperature for work spaces in the tropics. For this purpose the ordinary formula for the significance of difference between proportions was used,

$$\text{i.e. Standard error} = \sqrt{pq\left(\frac{1}{N_1} + \frac{1}{N_2}\right)}$$

A rise from 15·97 per cent. failures (the calculated incidence at 85°/75°) to 27·68 per cent. failures was needed for a statistically definite worsening of performance ($p = 0{\cdot}05$). This appeared to be a way of analysing the data which was rather less sensitive to the effects of heat and high humidity than the previous statistical technique because this placed the critical level at which W.T. reception definitely began to fall off at 90° F. on the effective temperature scale.

Absence of Day-to-Day Cumulative Effects

It might have been thought that since the temperature conditions were given in order of increasing severity the falling off in W.T. performance could have been attributed to some psychological changes accumulating from day to day. If that had been so, then the week-end break allowed Group B between the temperatures of 95°/85° and 100°/90° might have been expected to have abolished these cumulative effects, whereas, in fact, all these six men deteriorated after the break when faced with an effective temperature of 92° F. instead of 87·5° F.

Similarly, if cumulative effects had been marked, it might have been expected that the number of errors would have been higher the second day a particular room temperature was given. In fact, the subjects invariably did rather better on the second day of a particular room temperature. This was true of all the temperatures given (see Appendix I). Between effective temperatures of 79°–92° the average error score on the second day was usually about five-sixths of the score on the first day. With the highest atmospheric temperature of all, this slight but definite effect became even more marked. In other words, there was no evidence that the effects obtained were accumulating serially from day to day.

It is difficult to explain this improvement on the second day of a particular room temperature. It was not a simple learning or practice effect because

in addition to their extensive previous W.T. experience, the men in Group B were given about half a million letters and numbers before the main tests. Similarly, as all this training over a period of 7 weeks was given in the hot room, it seems unlikely that the subjects were still becoming more used to working in the heat. Lack of physiological acclimatization can also be excluded for the same reason, and also because this improvement on the second day of a particular room temperature was found at the quite moderate effective temperature of 79° F.

Effects of Long Watch Periods

A striking change was the rise in the number of errors within the 3-hour test period. The following table gives the average errors per man per hour (as estimated by arithmetic means) when each of the 3 hours of the test spell was considered in turn.

AVERAGE NUMBER OF ERRORS PER SUBJECT PER HOUR—ARITHMETIC MEANS

	Room temperature				
	85°/75° (79° ET)	90°/80° (83° ET)	95°/85° (87·5° ET)	100°/90° (92° ET)	105°/95° (97° ET)
1st hour	5·2 errors	5·0 errors	6·6 errors	7·9 errors	68·5 errors
2nd hour ..	11·2 errors	11·4 errors	13·0 errors	15·2 errors	32·9 errors
3rd hour	19·7 errors	18·3 errors	26·4 errors	29·1 errors	182·4 errors

It was interesting to see whether this form of deterioration was related to the level of W.T. operating efficiency.

AVERAGE NUMBER OF ERRORS PER SUBJECT PER HOUR—ARITHMETIC MEANS

Efficiency category		Room temperature				
		85°/75° (79° ET)	90°/80° (83° ET)	95°/85° (87·5° ET)	100°/90° (92° ET)	105°/95° (97° ET)
'Exceptionally skilled' group.	1st hour	1·3 errors	0·7 errors	2·2 errors	0·8 errors	3·3 errors
	2nd hour	2·5 errors	2·5 errors	1·5 errors	2·8 errors	5·8 errors
	3rd hour	5·0 errors	4·8 errors	3·8 errors	2·0 errors	9·3 errors
'Very good' group.	1st hour	2·3 errors	2·9 errors	3·1 errors	4·2 errors	14·6 errors
	2nd hour	4·9 errors	4·8 errors	6·2 errors	12·4 errors	10·4 errors
	3rd hour	11·3 errors	12·2 errors	14·4 errors	19·3 errors	274·0 errors
'Competent' group.	1st hour	13·8 errors	12·7 errors	17·0 errors	21·0 errors	223·7 errors
	2nd hour	30·5 errors	31·3 errors	36·0 errors	32·2 errors	97·5 errors
	3rd hour	48·3 errors	42·0 errors	69·0 errors	72·5 errors	202·0 errors

It will perhaps be more obvious from fig. 4 that with all three categories of operators there is a steady increase in errors as time passes during a 3-hour watch. The following table shows that the extent of this increase in errors is relatively greater

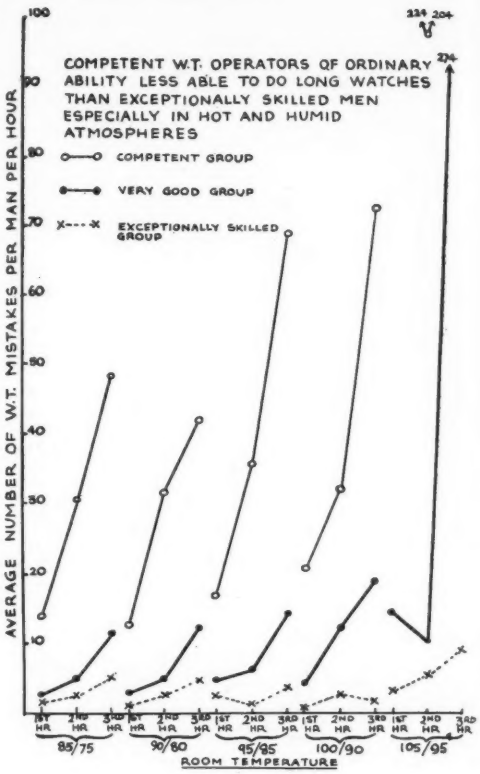

FIG. 4.

with the operators of average ability than with the exceptionally skilled men. The figures give the additional number of mistakes in the last hour of the test compared with the first hour for each efficiency category at each room temperature.

ADDITIONAL NUMBER OF MISTAKES PER SUBJECT PER HOUR IN THIRD HOUR OF WATCH COMPARED WITH FIRST HOUR

Efficiency category	Room temperature				
	85°/75° (79° ET)	90°/80° (83° ET)	95°/95° (87·5° ET)	100°/90° (92° ET)	105°/95° (97° ET)
'Exceptionally skilled' men.	+3·7 errors	+4·1 errors	+1·6 errors	+1·2 errors	+6·0 errors
'Very good' men.	+9·0 errors	+9·3 errors	+11·3 errors	+15·1 errors	+259·4 errors
'Competent' men.	+34·5 errors	+29·3 errors	+52·0 errors	+51·5 errors	−21·7 errors

It is interesting that the only exception to this increase in errors during the watch arose when the less efficient men were faced with the highest temperature of all and did so badly during the first hour that they could hardly have done much worse during the rest of the test period.

There were also signs that this deterioration with prolonged work was aggravated by having to work in high atmospheric temperatures.

Effects of Head Colds on Prolonged W.T. Reception

On two occasions (apart from the findings already described) records were made of performance when a subject was suffering from a severe head cold. Subject No. 2 made the following scores on one occasion when the effective temperature was 79° F.

SUBJECT NO. 2 WITH HEAD COLD—NUMBER OF ERRORS PER MESSAGE

1st message	2nd message	3rd message	4th message	5th message	6th message	7th message	8th message	9th message
0 errors	0 errors	2 errors	4 errors	10 errors	19 errors	24 errors	39 errors	349 errors

This is given in fig. 5 along with the four other available records previously made by No. 2 under the same room temperature conditions when he was feeling perfectly fit and well.

SUBJECT NO. 2 WHEN HEALTHY—NUMBER OF ERRORS PER MESSAGE

1st message	2nd message	3rd message	4th message	5th message	6th message	7th message	8th message	9th message
0	0	1	0	1	6	8	0	1
0	3	1	2	0	2	1	4	2
0	1	0	1	5	2	6	6	2
0	1	1	1	0	1	1	2	3

Ave rage Incidence

0 errors	1·2 errors	0·7 errors	1·0 errors	1·5 errors	2·7 errors	4·0 errors	3·0 errors	2·0 errors

Similarly, subject No. 8 under the 83° F. effective temperature conditions produced this record when suffering from a head cold.

SUBJECT NO. 8 WITH HEAD COLD—NUMBER OF ERRORS PER MESSAGE

1st message	2nd message	3rd message	4th message	5th message	6th message	7th message	8th message	9th message
2 errors	5 errors	1 errors	2 errors	0 errors	64 errors	93 errors	106 errors	106 errors

This was in striking contrast to seven previous or subsequent records made by No. 8 under the same atmospheric conditions when he was quite healthy.

SUBJECT NO. 8 WHEN HEALTHY—NUMBER OF ERRORS PER MESSAGE

1st message	2nd message	3rd message	4th message	5th message	6th message	7th message	8th message	9th message
0	2	0	1	2	0	4	9	6
4	1	3	0	3	0	2	0	0
1	2	1	5	2	1	3	4	4
1	1	1	2	1	3	1	1	2
1	5	1	1	2	5	8	10	9
0	1	0	3	3	1	5	3	0
3	3	1	1	1	0	4	9	5

Average Incidence

1·4 errors	2·1 errors	1·0 errors	1·8 errors	2·0 errors	1·4 errors	3·9 errors	5·1 errors	3·7 errors

There were no signs with either Subject No. 2 or No. 8 that the head cold had led to any abnormality of the rectal temperature.

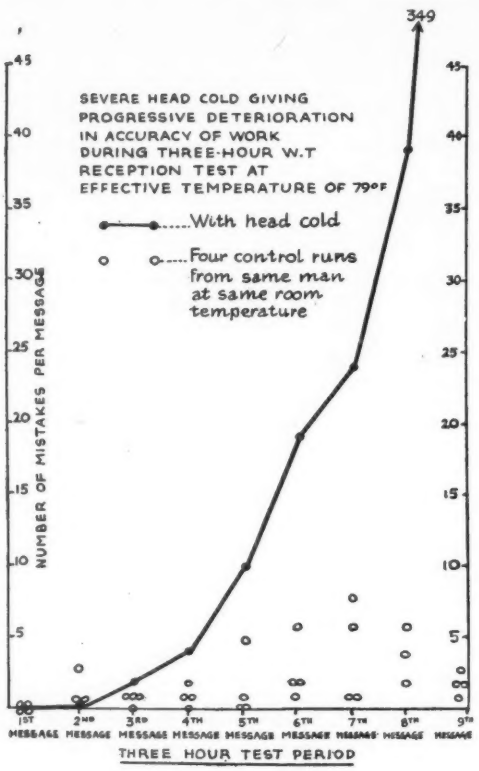
SEVERE HEAD COLD GIVING PROGRESSIVE DETERIORATION IN ACCURACY OF WORK DURING THREE-HOUR W.T RECEPTION TEST AT EFFECTIVE TEMPERATURE OF 79°F
With head cold
Four control runs from same man at same room temperature
NUMBER OF MISTAKES PER MESSAGE
349
1ST MESSAGE
2ND MESSAGE
3RD MESSAGE
4TH MESSAGE
5TH MESSAGE
6TH MESSAGE
7TH MESSAGE
8TH MESSAGE
9TH MESSAGE
THREE HOUR TEST PERIOD

FIG. 5.

2. Rectal Temperatures

Rectal temperature readings are available from each of these eleven men for each of the room temperatures. On two separate days for each room temperature the rectal temperature was measured before the W.T. reception test and then again 3 hours later at the end of the test. These readings are given in Appendix II.

The average rectal temperatures for the whole group of subjects when measured at the end of the W.T. test were clearly related to the room temperature and this finding is shown in fig. 6.

AVERAGE RECTAL TEMPERATURE AT END OF TEST SPELL

Room temperature	85°/75° (79° ET)	90°/80° (83° ET)	95°/85° (87·5° ET)	100°/90° (92° ET)	105°/95° (97° ET)
Average rectal temperatures at end of test spell.	98·45°	98·77°	99·02°	99·58°	100·49°

It will be seen that a rise in room temperature from 85°/75° to 90°/80° gives a rise of about a third of a degree in the body temperature. This is a statistically significant difference as $t=4{\cdot}2$ and the odds are therefore less than 1 in 100 that this is a chance finding. Similarly there is a further quarter degree rise in body temperature when the room temperature is raised from 90°/80° to 95°/85°. Thereafter the body temperature goes up rather more steeply because the increase in room temperature between 95°/85° and 100°/90° leads to a half degree rise, whereas the change between 100°/90° and 105°/95° gives a jump of one whole degree in the average body temperature.

Another method of analysing these findings was to calculate the rise or fall in the body temperature for each individual at each room temperature. In

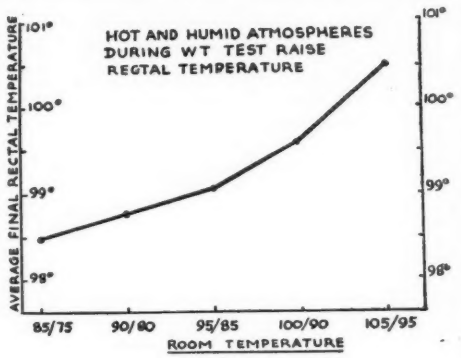

FIG. 6.

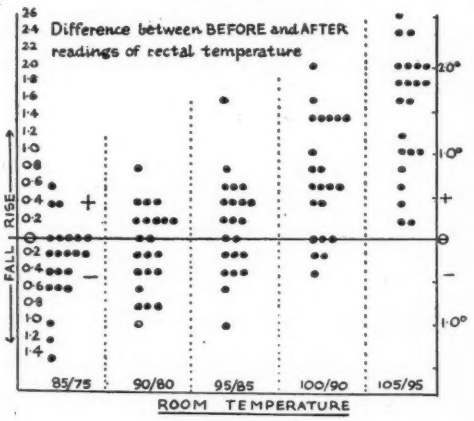

FIG. 7.

the 22 pairs of observations given in Appendix II for each room temperature the reading taken *before* the W.T. test was subtracted from the reading taken *after* the test (see fig. 7). A note was made of the number of times that the final body temperature was higher than the initial body temperature and the findings are given in the following table:

PROPORTION OF GROUP SHOWING RISE IN RECTAL TEMPERATURE DURING W.T. TEST

Room temperature	85°/75° (79° ET)	90°/80° (83° ET)	95°/85° (87·5° ET)	100°/90° (92° ET)	105°/95° (97° ET)
Incidence of rise in rectal temperature during W.T. test.	13·6% (3/22)	40·9% (9/22)	54·5% (12/22)	72·7% (16/22)	100·0% (22/22)

Similarly, at the various room temperatures the average difference in any temperature between the beginning and end of the test spell was:

Room temperature	85°/75° (79° ET)	90°/80° (83° ET)	95°/85° (87·5° ET)	100°/90° (92° ET)	105°/95° (97° ET)
Average difference in rectal temperature during test spell.	−0·28° F	−0·15° F	+0·14° F	+0·72° F	+1·47° F

3. Relationship between W.T. Errors, Rectal Temperatures and Atmospheric Temperature

Three kinds of observations have been made—physical, physiological and psychological. As both room temperatures and rectal temperatures can be measured much more readily than most types of human performance, it is of great interest to break down the relationship existing between these three sets of observations to see whether in fact it would have been sufficient to rely simply on the rectal temperatures alone to give an estimate of W.T. reception efficiency at various room temperatures.

At first this looks a promising approach, because if one takes only the average readings, an association is apparently found between the three sets of readings.

Room temperature (Dry/wet bulb)	85°/75° (79° ET)	90°/80° (83° ET)	95°/85° (87·5° ET)	100°/90° (92° ET)	105°/95° (97° ET)
Average rectal temperature at end of W.T. test.	98·45°	98·77°	99·02°	99·58°	100·49°
Average number of W.T. errors per man per hour.	12·0 errors	11·5 errors	15·3 errors	17·3 errors	94·7 errors

Hypothesis that raised Rectal Temperature causes W.T. Performance Deterioration

It is often assumed that if the body temperature is raised the working ability of the sedentary worker is bound to deteriorate and that raised rectal temperature will cause a falling off in working efficiency. What evidence is there for this assumption? When two factors appear to be closely associated, there is not necessarily a causal relationship between them since there may well be a third factor jointly influencing both factors. By analogy, a number of trees may be waving to and fro while some clouds speed across the sky, but few would suggest that the trees are brushing the clouds along.

Relationship between Body Temperature and W.T Performance when the Atmospheric Temperature Factor was held Constant

It was therefore necessary to study the relationship between rectal temperature and W.T. performance with the effect of the third factor of atmospheric temperature removed. All the observations made at a particular room temperature were divided into above average or below average for final rectal temperature and for W.T. reception errors. In other words, at any one room temperature each of the 17 attempts was placed into one of four categories depending on whether the rectal temperature was above or below average (rectal temperature + or −) and also on whether the number of W.T. errors was above or below average (W.T. errors + or −). The average used was always the median reading for the observations made *at a particular room temperature*. This ensured that the influence of this third factor, the room temperature, was being statistically removed. If it is true that raised rectal temperature causes W.T. errors, then one might still expect that the men with rectal temperatures above the average for the group would have also been those with more than the average number of W.T. errors, i.e. that the proportion of attempts in the square marked (x) would have been relatively high.

	(x)
+W.T. errors −Rectal temp.	+W.T. errors +Rectal temp.
−W.T. errors −Rectal temp.	−W.T. errors +Rectal temp.

The results of this analysis at each of the room temperatures taken in turn were as shown at foot of page.

(The ½ and ¼ readings arose from observations which themselves formed the average readings.)

When all these five sets were lumped together the results were:

+W.T. errors −Rectal temp. (28·5 per cent.)	24¼	18¼
(22·0 per cent.) −W.T. errors −Rectal remp.	18¾	23¾

		Room Temperature								
		85°/75° (79° ET)		90°/80° (83° ET)		95°/85° (87·5° ET)		100°/90° (92° ET)		105°/95° (97° ET)
+W.T. −R.T.	+W.T. +R.T.	3½	5¼	5	3½	5½	3	6	2½	4½	4
−W.T. −R.T.	−W.T. +R.T.	3¾	4¾	4½	4	3	5½	3	5½	4½	4

In the five sets of data taken from each of the room temperatures separately the chi-square readings were not significant—and for the combined data the chi-square was 1·4. In fact there was no relationship between rectal temperature and W.T. performance—*once the influence of the third factor of atmospheric temperature had been removed.* Consequently a rise in body temperature does *not* cause a rise in the number of W.T. errors.

Rectal Temperature Rise without Deterioration in W.T. Reception

Further evidence that a rise in rectal temperature does not lead to deterioration in W.T. reception efficiency is found in the following point. It will be remembered that on comparing average rectal temperature at a room temperature of 85°/75° with that obtained at 90°/80° there was a slight but definite rise. But there was no significant difference in the average accuracy of W.T. reception at the two room temperatures.

Hypothesis that Raised Rectal Temperature can predict W.T. Deterioration

A less ambitious version of the now discredited hypothesis given above might be to state that though a raised rectal temperature does not cause W.T. deterioration, nevertheless rectal temperature is probably a rough index of W.T. efficiency because apparently rectal temperature and the incidence of W.T. errors both depend on the third factor of atmospheric temperature.

Average W.T. Performance at Various Rectal Temperatures

To test this hypothesis, all the available body temperature readings taken at the end of the test spell were grouped into categories determined by class intervals of half a degree. Then the corresponding W.T. error scores were totalled and averaged for each of these different levels of rectal temperature as shown at foot of the page.

Only when the body temperature rose to 100·5°–101·1° was there any sign of a relationship between the two factors of rectal temperature and W.T. reception efficiency.

Individual Comparisons between W.T. Reception Efficiency and Rectal Temperature

It is possible that these average results were hiding complex relationships between the two factors; it seemed necessary therefore to consider the 85 individual instances in which the final rectal temperature for a subject could be plotted against the number of W.T. errors he made per hour (see fig. 8). Here again there were no signs of an association between individual performance at W.T. reception and rectal temperature except with the rectal temperatures above 100·5° F. which without exception were obtained in the 105°/95° room temperature, i.e. atmospheric conditions which were more severe than those found in the Eastern Fleet (Ellis, 1946).

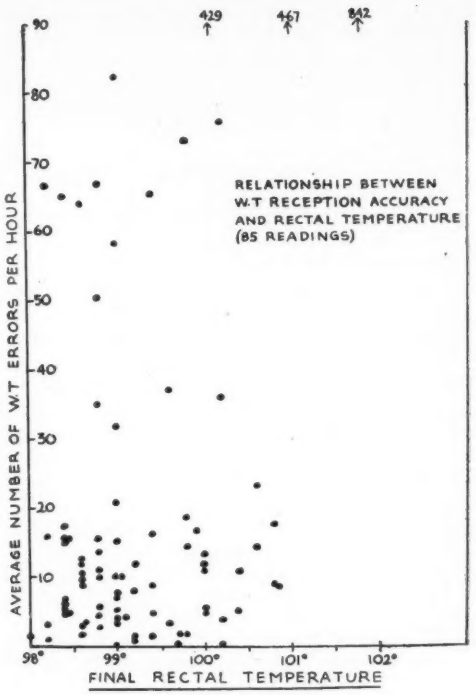

FIG. 8.

Reasons for Failure of Individual Body Temperature and W.T. Performance to correlate once effects of Atmospheric Temperature have been removed

The reasons for this failure of the body temperature to give even a rough index of possible W.T. performance are clearly the following facts:

(*a*) Quite apart from the effects of heat, different people have entirely different levels of ability at W.T. reception—and these are not related to body temperature.

To be sure that these individual differences in ability at W.T. reception were not the sole reason for the lack of correlation, a comparison was made between final rectal temperature and the difference between the number of W.T. errors made during the first hour of the test spell and the last hour.

Rectal temperature category	98·0° F. –98·4° F.	98·5° F. –98·9° F.	99·0° F. –99·4° F.	99·5° F. –99·9° F.	100·0° F. –100·4° F.	100·5°F. –101·1° F.
Average number of mistakes per message (median readings).	1·0 errors	2·0 errors	1·0 errors	2·0 errors	1·5 errors	5·0 errors

There was no great improvement on the correlation shown in fig. 8.

(*b*) The extent of any deterioration in W.T. performance under conditions of heat and high humidity is partly determined by the level of proficiency of the operator.

(*c*) Progressive deterioration during the test spell arising from prolonged work at W.T. reception cannot be measured by body temperature. A glance at fig. 4 and fig. 7 shows that during the W.T. test given at a room temperature of 85°/75° the rectal temperature of most of the subjects was either falling or quite unchanged while the W.T. errors were usually increasing.

(*d*) Individual differences in rectal temperature can be quite marked even among healthy men, e.g. subject No. 6 often had a rectal temperature of about 100° F. under normal circumstances. When, however, the rise or fall in rectal temperature was substituted for the absolute reading at the end of the test, no appreciable improvement was noted in the degree of association between the two variables. This was true whether the W.T. reception scores were considered as the average error score during the whole 3 hours, the rise or fall in W.T. errors during test spell or the W.T. errors made in the last hour. These results do not agree with those of Kleitman (1939) who noted a tendency towards deterioration in reaction time with a fall [*sic*] of even a fraction of a degree in mouth temperature.

Conclusions

1. When the room temperature was raised to dry bulb/wet bulb readings of 95°/85° there was a statistically definite fall in accuracy of W.T. reception compared with performance at room temperatures of either 85°/75° or 90°/80°. Since the air velocity was 100 feet per minute, this means that *effective temperatures of* 79° *F. to* 83° *F. were undoubtedly leading to better W.T. reception than an effective temperature of* 87·5° *F.* In the First Report of the Habitability Mission to the Eastern Fleet it was noted that the majority of W.T. ratings in the Eastern Fleet had to work at effective temperatures which were above 84° F. and in prolonged action the conditions were even more severe.

2. On an entirely different form of work in which the task was to move a heavy lever with precision the critical point on the room temperature scale also lay between the effective temperatures of 83° F. and 87·5° F. The average number of W.T. mistakes proved to be a more sensitive measure than the incidence of faulty W.T. messages.

3. Though all the men tested were sufficiently experienced at W.T. reception to have been in important operational positions, the exceptionally able members of the group were less affected by the harassing atmospheric conditions than men regarded as very good operators. Similarly, men of ordinary ability showed the greatest deterioration of all. In other words the better men stood up more easily to the difficult conditions.

4. The deterioration as measured by the number of faulty W.T. messages bore a logarithmic relationship to the atmospheric temperature changes. The curve had the general formula

$$\log Y = c_0 + c_1 x$$

when c_0 and c_1 were constants, x room temperature and Y the proportion of failures. The same general formula also applied when the nature of the task was quite different, and consisted simply of moving a heavy lever with speed and precision.

5. There were no signs of psychological effects accumulating serially from day to day; in fact the men were usually rather better at the W.T. test on the second day of a particular room temperature. It is not clear exactly why that happened.

6. The average number of W.T. errors per man per hour usually rose considerably and progressively within the 3-hour watch period. In general, twice as many errors were made in the second hour and three or four times as many during the third hour. Here again, however, the deterioration was more marked in operators of average ability than in the exceptionally skilled.

7. Severe head colds greatly increased this falling-off during the 3-hour spell. It seems likely that this was due to the accompanying discomfort rather than to any temporary deafness, since the men began their work with perfect or almost perfect accuracy.

8. Rectal temperature responded readily to changes in the atmospheric temperature and was in fact a most sensitive measure of atmospheric conditions.

9. Rectal temperature failed, however, to estimate the psychological effects of these atmospheric conditions.

10. There seem to be five reasons why rectal temperature did not predict the effects of hot and humid atmospheres on accuracy of W.T. reception.

(i) Even trained men differed considerably among themselves in their W.T. reception ability.

(ii) The extent of their deterioration in accuracy in the heat also varied from one man to another, the better operators stood up more easily to the heat.

(iii) The extent of the loss of accuracy arising from prolonged spells of W.T. reception was also partly determined by the level of operating ability.

(iv) Persistent individual differences in rectal temperature were found in different people. During the day the normal rectal temperature may also vary by as much as 1° F.

(v) Various attempts to deal statistically with these causes of dissociation between the two variables showed that the main reason for the lack of correlation between rectal temperature and W.T. performance score was simply that raising the temperature of the body did not lead to deterioration in W.T. performance until rectal temperatures of 100·5° F. were obtained. Only in 8 per cent. of the observations were rectal temperatures as high as this obtained at the end of the W.T. test and all these were found at the worst room temperature of all, a dry bulb/wet bulb reading of 105/95, which was even more severe than the atmospheric conditions encountered by W.T. operators in the Eastern Fleet.

Acknowledgments

I am greatly indebted to Professor F. C. Bartlett, F.R.S. and Mr. E. G. Chambers, M.A., of this laboratory for much advice and help during this investigation which has been done for the Medical Research Council and was undertaken at the request of the Habitability Sub-Committee of the Royal Naval Personnel Research Committee. It followed a visit to H.M.S. *King George V* with Surgeon-Captain Macdonald Critchley, R.N.V.R., to gain first-hand information on naval duties.

The Medical Departments of the Royal Navy and Royal Air Force supplied the Wireless Operators from H.M.S. *Mercury* and No. 26 Group, R.A.F.; most of the W.T. equipment was provided by Air Vice-Marshal O. G. W. G. Lywood, A.O.C., No. 26 Group, R.A.F. ; the rest came from Dr. G. A. Knight, of the Department of Scientific Research and Experiment, Admiralty.

The illustrative diagrams in the report were prepared by Sergeant S. Nixon, W.A.A.F. Corporal Holloway, R.A.F., was responsible for the maintenance of the technical equipment and the actual experimental testing was done by L.A.C. J. H. Bonner and L.A.C. C. A. Hare, R.A.F.

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APPENDIX I

DATA FROM W.T. RECEPTION TESTS

TABLE 1

ATMOSPHERIC TEMPERATURE AT 85°/75° (EFFECTIVE TEMPERATURE 79° F.)

Subject's index number, efficiency category and length of previous W.T. experience	Number of mistakes per hour			Total number of mistakes in 85°/75° for each test	Average number of mistakes per hour in 85°/75° for each test	Average number of mistakes per hour in 85°/75° for each subject	Average number of mistakes per hour in 85°/75° for each efficiency category
	1st hour	2nd hour	3rd hour				
'Exceptionally skilled' group—							
Subject 1 (25: 3 years W.T.).	3	1	1	5	1·7	1·7	
Subject 2 (29: 3 years W.T.).	1	2	1	4	1·3	1·3	
Subject 3 (23: 3 years W.T.).	0	4	17	21	7·0	5·8	
	0	5	9	14	4·7		2·9
'Very good' group—							
Subject 4 (31: 3 years W.T.).	1	0	8	9	3·0	3·0	
Subject 5 (34: 3 years W.T.).	5	13	12	30	10·0	10·0	
Subject 6 (23: 4 years W.T.).	2	0	11	13	4·3	4·3	
Subject 7 (32: 3 years W.T.).	1	7	9	17	5·7	4·5	
	2	2	6	10	3·3		
Subject 8 (19: 1 year W.T.).	3	9	27	39	13·0	9·0	
	1	5	9	15	5·0		6·2
'Competent' group—							
Subject 9 (18: 1 year W.T.).	6	11	30	47	15·7	12·0	
	1	8	16	25	8·3		
Subject 10 (29: 3 years W.T.).	5	7	38	50	16·7	14·5	
	1	12	24	37	12·3		
Subject 11 (26: 3 years W.T.).	33	69	94	196	65·3	66·2	
	37	76	88	201	67·0		30·9

APPENDIX I (*continued*)

TABLE 2

ATMOSPHERIC TEMPERATURE AT 90°/80° (EFFECTIVE TEMPERATURE 83° F.)

Subject's index number and efficiency category	Number of mistakes per hour			Total number of mistakes in 90°/80° for each test	Average number per hour in 90°/80° for each test	Average number per hour in 90°/80° for each subject	Average number of mistakes per hour in 90°/80° for each efficiency category
	1st hour	2nd hour	3rd hour				
'Exceptionally skilled' group—							
Subject 1	0	0	2	2	0·7	0·7	
Subject 2	0	4	7	11	3·7	3·7	
Subject 3	0	5	7	12	4·0	3·7	
	4	2	4	10	3·3		2·7
'Very good' group—							
Subject 4	3	1	17	21	7·0	7·0	
Subject 5	0	1	6	7	2·3	2·3	
Subject 6	4	11	10	25	8·3	8·3	
Subject 7	5	10	19	34	11·3	9·3	
	0	6	16	22	7·3		
Subject 8	2	3	19	24	8·0	6·1	
	8	3	2	13	4·3		6·6
'Competent' group—							
Subject 9	7	23	12	42	14·0	12·3	
	6	6	20	32	10·7		
Subject 10	0	7	42	49	16·3	16·1	
	5	21	22	48	16·0		
Subject 11	38	64	91	193	64·3	57·5	
	20	67	65	152	50·7		28·7

TABLE 3

ATMOSPHERIC TEMPERATURE AT 95°/85° (EFFECTIVE TEMPERATURE 87·5° F.)

Subject's index number and efficiency category	Number of mistakes per hour			Total number of mistakes in 95°/85° for each test	Average number of mistakes per hour in 95°/85° for each test	Average number of mistakes per hour in 95°/85° for each subject	Average number of mistakes per hour in 95°/85° for each efficiency category
	1st hour	2nd hour	3rd hour				
'Exceptionally skilled' group—							
Subject 1	0	0	4	4	1·3	1·3	
Subject 2	1	0	0	1	0·3	0·3	
Subject 3	6	5	14	25	8·3	5·8	
	5	4	1	10	3·3		2·5
'Very good' group—							
Subject 4	1	10	35	46	15·3	15·3	
Subject 5	8	2	8	18	6·0	6·0	
Subject 6	0	0	7	7	2·3	2·3	
Subject 7	2	10	5	17	5·7	8·0	
	2	12	17	31	10·3		
Subject 8	2	2	11	15	5·0	7·8	
	7	14	11	32	10·7		7·9
'Competent' group—							
Subject 9	9	5	18	32	10·7	13·3	
	12	14	22	48	16·0		
Subject 10	6	28	142	176	58·7	38·2	
	5	17	31	53	17·7		
Subject 11	29	83	109	221	73·7	70·5	
	41	69	92	202	67·3		40·7

APPENDIX I (*continued*)

TABLE 4

ATMOSPHERIC TEMPERATURE AT 100°/90° (EFFECTIVE TEMPERATURE 92° F.)

Subject's index number, efficiency category and length of previous W.T. experience	Number of mistakes per hour			Total number of mistakes in 100°/90° for each test	Average number of mistakes per hour in 100°/90° for each test	Average number of mistakex per hour in 100°/90° for each subject	Average number of mistakes per hour in 105°/95° for each efficiency category
	1st hour	2nd hour	3rd hour				
'Exceptionally skilled' group—							
Subject 1	0	2	0	2	0·7	0·7	
Subject 2	0	4	2	6	2·0	2·0	
Subject 3	2	3	6	11	3·7	3·0	
	3	2	2	7	2·3		1·9
'Very good' group—							
Subject 4	10	28	12	50	16·7	17·6	
Subject 5	4	9	24	37	12·3	12·3	
Subject 6	2	1	0	3	1·0	1·0	
Subject 7	1	15	36	52	17·3	18·0	
	3	22	31	56	18·7		
Subject 8	1	5	28	34	11·3	11·8	
	5	6	26	37	12·3		12·0
'Competent' group—							
Subject 9	9	21	34	64	21·3	17·3	
	5	6	29	40	13·3		
Subject 10	5	5	86	96	32·0	33·6	
	22	19	65	106	35·3		
Subject 11	53	76	120	249	83·0	74·6	
	32	66	101	199	66·3		41·8

TABLE 5

ATMOSPHERIC TEMPERATURE AT 105°/95° (EFFECTIVE TEMPERATURE 97° F.)

Subject's index number, efficiency category and length of previous W.T. experience	Number of mistakes per hour			Total number of mistakes in 105°/95° for each test	Average number of mistakes per hour in 105°/95° for each test	Average number of mistakes per hour in 105°/95° for each subject	Average number of mistakes per hour in 105°/95° for each efficiency category
	1st hour	2nd hour	3rd hour				
'Exceptionally skilled' group—							
Subject 1	3	6	18	27	9·0	9·0	
Subject 2	5	7	3	15	5·0	5·0	
Subject 3	2	7	7	16	5·3	4·5	
	2	2	7	11	3·7		6·2
'Very good' group—							
Subject 4	37	16	59	112	37·3	37·3	
Subject 5	13	10	21	44	14·7	14·7	
Subject 6	14	6	6	26	8·7	8·7	
Subject 7	9	10	34	53	17·7	11·5	
	5	1	10	16	5·3		
Subject 8	2	12	2513	2527	842·3	426·8	
	2	17	15	34	11·3		99·8
'Competent' group—							
Subject 9	15	22	33	70	23·3	18·8	
	12	13	18	43	14·3		
Subject 10	14	39	176	229	76·3	254·1	
	12	406	878	1296	432·0		
Subject 11	1266	60	76	1402	467·3	251·8	
	23	45	41	109	36·3		174·9

APPENDIX II

DATA FROM RECTAL TEMPERATURE MEASUREMENTS

Subject Nos.	Atmospheric temperature 85°/75° (Effective temperature 79°)			Atmospheric temperature 90°/80° (Effective temperature 83°)			Atmospheric temperature 95°/85° (Effective temperature 87·5°)			Atmospheric temperature 100°/90° (Effective temperature 92°)			Atmospheric temperature 105°/95° (Effective temperature 97°)		
	Before	After	Difference	Before	After	Difference	Before	After	Difference	Before	After	Difference	Before	After	Difference
Subject No. 1 ..	99·4	98·4	−1·0	100·4	99·2	−1·2	99·0	98·6	−0·4	99·6	99·6	0	100·2	100·8	+0·6
	99·4	98·0	−1·4	99·6	99·2	−0·4	99·4	99·2	−0·2	99·8	99·6	−0·2	99·8	100·8	+1·0
Subject No. 2 ..	98·8	98·2	−0·6	99·0	98·6	−0·4	99·0	99·0	0	98·8	99·4	+0·6	100·2	100·4	+0·2
	98·6	98·2	−0·4	99·0	98·6	−0·4	99·2	99·0	−0·2	99·0	99·4	+0·4	99·2	100·0	+0·8
Subject No. 3 ..	98·6	98·4	−0·2	98·6	99·0	+0·4	98·6	99·2	+0·6	98·6	99·6	+1·0	98·4	100·4	+2·0
	98·4	98·4	0	99·0	98·2	−0·8	98·4	99·0	+0·6	98·4	99·8	+1·4	98·4	100·2	+1·8
Subject No. 4 ..	98·8	98·2	−0·6	99·0	98·8	−0·2	99·0	99·2	+0·2	99·6	99·6	0	99·2	100·2	+1·0
	99·0	98·8	−0·2	99·0	98·4	−0·6	99·9	99·0	−0·9	99·8	99·4	−0·4	99·2	99·6	+0·4
Subject No. 5 ..	98·4	98·4	0	98·4	98·6	+0·2	98·4	98·6	+0·2	98·2	99·6	+1·4	98·8	100·0	+1·2
	98·6	98·6	0	98·4	98·6	+0·2	99·2	98·8	−0·4	99·2	99·2	0	99·6	99·8	+0·2
Subject No. 6 ..	99·4	98·8	−0·6	100·0	99·2	−0·8	100·0	99·6	−0·4	99·7	100·4	+0·7	100·0	102·0	+2·0
	100·2	99·1	−1·1	100·3	99·4	−0·9	100·4	99·7	−0·7	100·4	100·2	−0·2	99·8	100·8	+1·0
Subject No. 7 ..	98·4	98·4	0	98·8	98·8	0	98·6	99·0	+0·4	98·0	99·9	+1·9	98·4	100·8	+2·4
	98·8	98·6	−0·2	98·6	99·0	+0·4	98·4	98·8	+0·4	98·4	99·8	+1·4	98·4	100·0	+1·6
Subject No. 8 ..	98·6	98·4	−0·2	98·8	99·0	+0·2	98·8	99·4	+0·6	98·6	100·0	+1·4	99·2	101·8	+2·6
	98·8	98·4	−0·4	99·0	98·8	−0·2	98·6	99·0	+0·4	98·6	100·0	+1·4	98·6	100·4	+1·8
Subject No. 9 ..	98·8	98·4	−0·4	98·8	98·8	0	98·6	99·0	+0·4	98·4	99·0	+0·6	98·6	100·6	+2·0
	98·6	98·6	0	98·4	98·6	+0·2	98·6	98·4	−0·2	98·4	100·0	+1·6	98·2	100·6	+2·4
Subject No. 10 ..	98·0	98·4	+0·4	98·4	98·2	−0·2	98·2	99·0	+0·8	98·4	99·0	+0·6	98·4	100·2	+1·8
	98·0	98·6	+0·6	98·4	98·8	+0·4	98·4	98·4	0	98·4	98·8	+0·4	98·4	100·1	+1·7
Subject No. 11 ..	98·0	98·4	+0·4	98·4	98·6	+0·2	98·2	99·8	+1·6	98·2	99·0	+0·8	99·0	101·0	+2·0
	98·4	98·2	−0·2	98·0	98·8	+0·8	98·6	98·8	+0·2	98·6	99·4	+0·8	98·4	100·2	+1·8
Averages ..	98·73	98·45	−0·28	98·92	98·77	−0·15	98·88	99·02	+0·14	98·86	99·58	+0·72	99·02	100·49	+1·47

APPENDIX III

DESCRIPTION OF EXPERIMENTAL ARRANGEMENTS

Forty-five different messages were prepared from a standard series of training drills. Each message was composed of 250 groups of five-character syko, i.e. random mixed letters and numbers in the proportion of 2 : 1. An example of the nature of the material would be ..... J49Y4 B8C9M 126T6 B7LUB FOW52. A standard preamble (vvvvvvvvvv GFXvGFZ NRXIA GR250) preceded each message.

Each message was punched on a long paper tape by means of a Creed perforating machine, this being done to avoid errors in the transmitted message and to ensure constant transmission speed. At the appropriate time this tape was fed through a G.N.T. Wheatstone Transmitter Model 112 (A.M. 10G/2383) the speed of which was controlled at 22 words per minute by a G.N.T. Speed Regulator Model 2042, A.M. 10G/3242 (see fig. 9). The holes punched in the moving paper tape produced a series of electrical contacts which were converted into auditory morse by a Type No. 2 Valve Buzzer (A.M. 10A/10158), the note selector of which was set at position 3 to give a 1000 cycle sound. The whole of this transmitting apparatus was installed outside the hot room for convenience of operation; the output leads from the valve buzzer were taken to a terminal block inside the room.

The subjects were given a definite position at the table in the room (see fig. 1) and kept this place for every test. Their headphones were clearly labelled to ensure that each man always used his own pair. The volume in the headphones was constant and has been estimated at 50–60 phons.

Each written answer sheet was checked three times, twice by W.T. operators other than the subject, the third and final check being made by the subject himself.

FIG. 9

A METHOD FOR STUDYING VIBRATIONS TRANSMITTED TO THE HANDS

BY

JOHN N. AGATE and H. A. DRUETT

From the Department for Research in Industrial Medicine (Medical Research Council), The London Hospital.

There is an increasing tendency in engineering practice to put into the hands of workers power-driven tools of all kinds for work which involves hammering, chiselling, grinding and polishing. The speed and efficiency of working is increased and the physical labour involved is lessened, but most of these portable tools cause vibration to be transmitted direct to the hands. Studies completed in the past 30 years, and those now in progress, attribute clinical effects to the use of such tools, and the factor common to them all is the vibration set up. Middleton (1930) reported the Raynaud phenomenon in those who operated pounding-up machines in the boot and shoe trade, but these machines have since passed out of use. The same syndrome, variously called 'white fingers,' 'dead fingers,' or 'dead hand,' has since been described frequently in the literature. Maria Seyring (1930) reported it in those who were fettling iron castings with pneumatic tools. Hunt (1936) also observing pneumatic tool workers, pointed out that the hand which was worst involved was the one which appeared to be subjected to the most vibration, that is, the hand in front which grasps the tool at its striking end. Other cases were recorded by Teleky (1927), McLaren (1937), Bennett, Waine and Bauer (1942), Barker and Hines (1944), Lindquist and Flemberg (1945), and Hunter, McLaughlin and Perry (1945). The latter studied 286 men who had worked with pneumatic tools of various sizes, and in a variety of occupations. White fingers were noted in 161, and developed usually after 2–5 years of such work. As in all the foregoing examples cold was the principal precipitating factor. In 148 cases in this series there were small circumscribed areas of decalcification demonstrable in radiographs of the carpal bones, a fact which had previously been noted by Brailsford (1934) (1936) and McLaren (1937), though the appearance is certainly not to be found solely in pneumatic tool workers. The incidence of arthritis of the elbow and wrist joints and of injury to the ulnar nerve or fasciae of the palm was not high in this series, though there have been references to these conditions since 1930.

In the war years there has been a rapid increase of work involving the polishing of steel castings and scurfing of aluminium alloy castings used in the manufacture of aero engines. The practice favoured at the time of writing is for a hand tool to be used to facilitate work in awkward corners and within the castings. The tool consists of a hand grip attached to a flexible cable drive running from an electric motor suspended from the roof. Various polishing, grinding and cutting heads can be used in this tool, and further cases of Raynaud's phenomenon have resulted, due to the intense vibration set up in the hands (Telford, McCann and MacCormack (1945)). Other cases are known to be occurring in industries where metal components are held in the fingers for grinding against grinding-wheels on fixed mountings, the vibration in this case being transmitted not from the machine but from the article being ground.

It has been suggested that various other factors assist the appearance of clinical effects from the use of vibrating tools. The current of cold exhaust air from pneumatic hammers is one, the hardness of the material on which the men use their tools is another. But the rates of rotation of the grinding tools or of the hammer strokes in pneumatic tools have been particularly considered. Cummings (1940) and Mills (1942) suggest that hammer strokes of 2500 per minute are more likely to cause Raynaud's phenomenon than other speeds, and in the cases of Hunter, McLaughlin and Perry a higher incidence was noted in those who worked with tools at 2000 to 3000 strokes per minute than at speeds above or below. The issue is, however, complicated by the fact that on the softer metals such as hot rivet heads, a slower type of hammer is used, and the decreased incidence of the phenomenon might be from either cause. The heaviest pneumatic tools, besides being the slowest, are usually used pointing downwards, and the work is done as much by the tools' weight as by the efforts of the workman. Similarly, the fastest tools are used on the lightest jobs, so that the grip and exertions of the operator can be less, and he probably absorbs less vibration in consequence. Rotating polishing tools are usually used at about 1900–4000 r.p.m., but light compressed air turbine tools for fine work may work at above 30,000 r.p.m., and in these the vibrations set up are apparently of a finer type, and the operator's grip on the tool is light.

No great advance towards discovering the cause of the clinical effects of vibrations, or of obviating them, is likely to be possible until a method for accurately recording their nature has been evolved. When this is done, it might be possible by correlating clinical and physical data to determine whether there is a critical frequency likely to cause the

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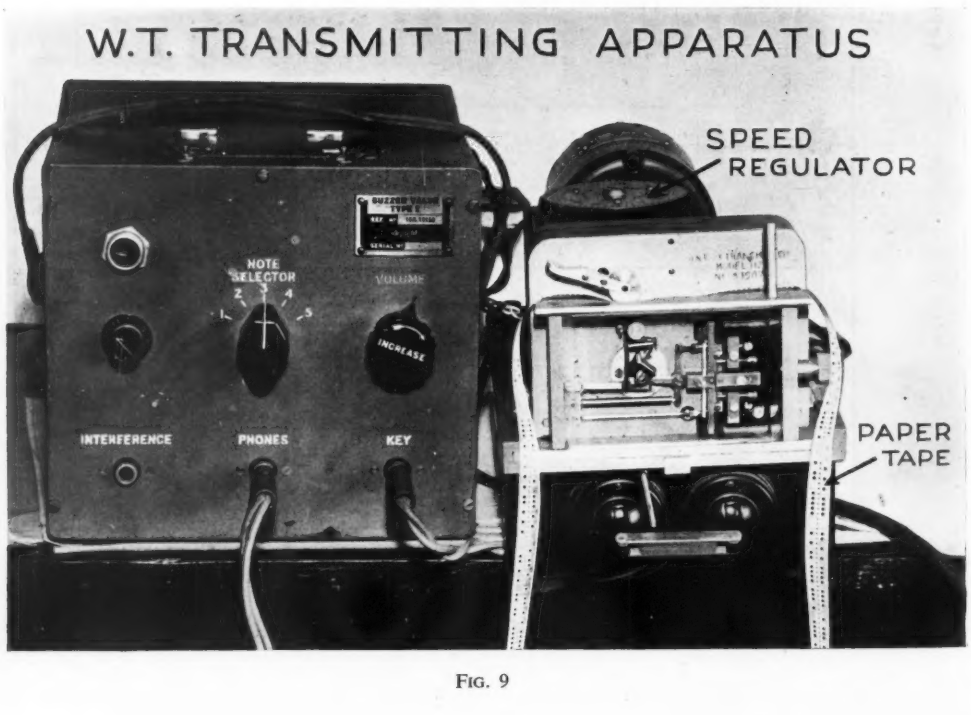

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No great advance towards discovering the cause of the clinical effects of vibrations, or of obviating them, is likely to be possible until a method for accurately recording their nature has been evolved. When this is done, it might be possible by correlating clinical and physical data to determine whether there is a critical frequency likely to cause the

syndrome; secondly, it might be possible to predict the chances of a given tool producing it, and thirdly, the efficacy of countermeasures such as the use of absorbent hand grips and pads could be studied. But the vibration set up by any of these tools is not of a single frequency nor has it a curve of simple sine-wave type. There may be a fundamental of low frequency, but, as in the case of a musical instrument, there are harmonics of higher frequency overlying it. In a rotating machine the fundamental bears a relation to the shaft speed, and the harmonics which modify it are provided by the irregularities of the shaft and its bearings and by such factors as the number of teeth on the cutting head, while in an oscillating pneumatic hammer the fundamental relates to the to-and-fro action of the internal piston, though the natural resonance of the metal of which it is made sets up a secondary set of high-frequency vibrations. In both types the natural resonance of the materials worked upon is transmitted back into the tool and thence to the workman's hands, while the power of his grip and the weight of his arms modify the vibrations. What is more, the vibration of the tool while idling differs from that while it is working, and the frequencies alter from time to time as the man presses harder to grind and so slows down his machine.

Fourier Analysis

When harmonics are superimposed on a fundamental vibration they modify its waveform, as depicted graphically, from the simple sine-wave curve. In the vibrations of machinery, even more than in simple instrumental notes the waveform becomes exceedingly complex. Fourier at the beginning of the last century showed that any uniformly repetitive phenomenon, however complicated its waveform, could be resolved as the sum of a series of waves of the sine type, the frequencies of the component waves being simple multiples of that of the main wave under consideration. These higher frequency waves are its harmonics. In the present investigation an electrical Fourier analysis has been made on the composite vibrations, to break them up into their constituent harmonics and determine the frequency and amplitude of each.

If it can be assumed that the local pathological effects of vibrations in the hands are related in some way to the quantity of energy absorbed by the tissues, it is unreasonable to assume that the effects of energy represented by the low-frequency components are necessarily the same as those represented by the high frequency. Already there are being noted bizarre biological effects from very high-frequency vibrations outside the audible range, otherwise known as 'supersonic' vibrations. In the same way the biological effects of high-frequency alternating currents differ widely from those of low frequency. It is by no means proven, though it is likely, that the pathological process set in action by the vibrations takes place entirely locally in the palm of the hand or the digits; arteriograms have shown spasm occuring locally in the peripheral vessels in the hand, and this may be the case in other examples of Raynaud's phenomenon of different aetiology. The question will probably remain unsolved until there is histological evidence taken from the vessels in the hand of a pneumatic tool worker. The opportunities for such a study are naturally very infrequent.

The requirements of any method for studying the problem experimentally are therefore that it shall make possible the analysis of the total vibration taking place at any given moment in terms of amplitudes and frequencies, which may lie between 20 and 10,000 vibrations per second or above. The time taken over each analysis must be short or the conditions of the experiment may have changed meanwhile. The apparatus must be sufficiently portable to enable it to be taken to the worker at his job, for most of the tools and materials worked upon are too bulky to be studied in the laboratory. Whatever the method of picking up the vibrations, it must not hinder the use of the workman's hands in any position, nor must his normal grip be materially altered. Mechanical or fluid transmission from the vibrating surfaces to any recording apparatus is clearly useless both because of the inertia of any such system, and because of its impracticability on all of the above counts. An electrical method has therefore suggested itself.

The Piezoelectric Crystal

Certain crystals which have no centre of symmetry, such as quartz, tourmaline, or Rochelle salt, exhibit piezoelectric effect, that is, when distorted by pressure they develop an electric potential, and, as discovered by the Curies, the potential generated is proportional to the pressure applied. A crystal suitably mounted can equally well be distorted by being shaken, and therefore can be used to pick up vibrations. Rochelle salt (sodium potassium tartrate) crystals have 1000 times the piezoelectric effect of quartz, and can be grown artificially. Variations in the response of individual crystals, particularly in response to changes of temperature, are minimized by making laminated or 'bimorphic' crystals of two plates bonded together. Such crystals will accurately follow vibrations at least up to 10,000 cycles, and their electrical response can be analysed by methods to be described below. The crystal pickups used in our experiments were of Rochelle salt and were supplied by Messrs. Rothermel, Ltd. They were fixed in mountings measuring $25 \times 32 \times 4$ mm. The crystal projected out of one end, and was otherwise covered by a metal housing; at the other end was fixed the screened lead that conducts the response. In the early stages the crystal was strapped directly to the tool under investigation, but it was found to become damaged by handling and pressure, and in particular by the heat generated during certain of the grinding work, for the piezoelectric properties of crystals are permanently lost after exposure to 130° F. It was therefore necessary to make a

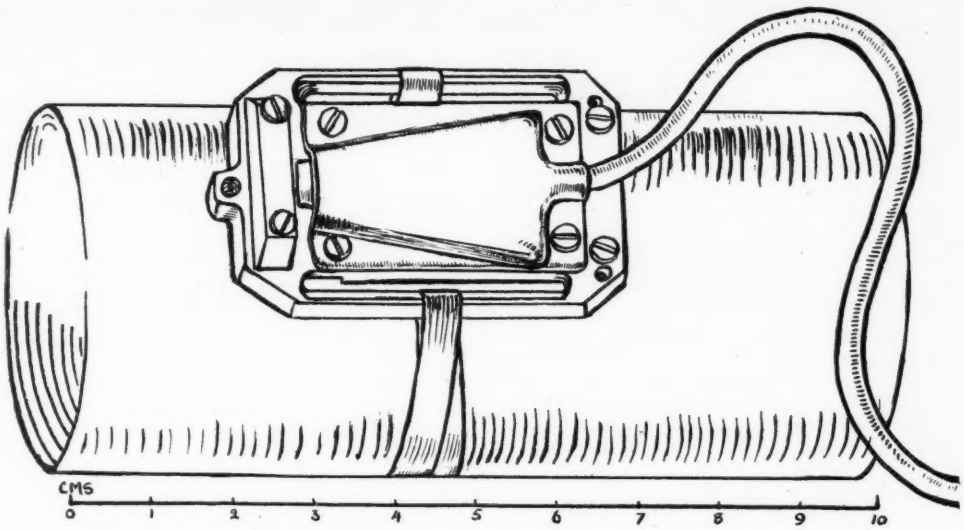

FIG. 1.—The piezoelectric crystal and mounting, shown strapped to a cylinder of metal. The pointed set-screws which make direct contact are hidden.

new mounting. The crystal in its original cover was screwed to a metal base-plate (fig. 1) to reduce the pressure distortion and the risk of damage by heat, the only disadvantage being a slight increase in bulk, though the pickup would still lie comfortably in the palm of the hand. The base-plate had a protective bar at one end to guard the naked point of the crystal and a slot along each side by which it could be tightly bound by inelastic tapes to the handle of any tool. Direct metallic contact was ensured by four pointed and adjustable steel set-screws which allowed the pickup to rest on a surface of any curvature, and which could be withdrawn if readings were to be taken from skin or any other soft surface. As a rule the pickup was attached to the handle of a tool at the point where it is usually held, and the man's hand would lie on top of it, the disturbance to his normal grip being negligible and the limitation of his movements solely that imposed by the length of the pickup lead. The base-plate was designed also so that it could be clamped when desired to one arm of an electrically maintained tuning fork. The amplitude of the vibration at the point of clamping was measured optically with a microscope having a micrometer eye-piece calibrated against a stage micrometer, a point on the crystal itself being observed under horizontal illumination as it moved to and fro. The response of the pickup to a standard deflection could by this means be checked from time to time: this was found particularly necessary as the crystal had to be subjected to rough handling during normal working conditions.

Analysis of the Pickup Response

In theory the voltage generated in the crystal pickup could be made to displace the electron beam of a cathode-ray oscillograph and a picture of the waveform would appear on the fluorescent screen. From this the constituent harmonics might be calculated. In practice, while this picture is relatively easy to obtain, its interpretation in terms of the harmonics is at best difficult and inaccurate. Usually it is impossible. The transience and the variable nature of the vibrations from machines do not simplify the matter.

To avoid the difficulties use has to be made of the properties of electrical filter circuits through which the pickup response can be passed. A 'high-pass filter' will entirely reject frequencies below a certain chosen value and pass those above. A 'low-pass filter' will pass frequencies below a certain value and reject those above. A 'band-pass filter' will pass frequencies between two chosen values and reject those above and below. This band in the latter type can be made almost narrow enough to select a single frequency alone, and this principle is embodied in the instrument first used in these experiments, a Harmonic Wave Analyser to the specification of Messrs. Marconi Instruments, Ltd. This apparatus was a battery model and portable, besides being sensitive and capable of defining frequencies within narrow limits. It could be tuned on a graduated dial to any frequency in the range under investigation, and the proportion of the crystal response corresponding to that frequency measured on a millivoltmeter. In operating the instrument the tuning dial was turned slowly and the points of maximum deflection of the meter needle were observed and the corresponding reading in cycles per second on the dial noted. The meter registered throughout, and the 'peaks' of its deflection had to be determined to place the principal frequencies. Graphs could then be constructed

by plotting output in millivolts and frequency as ordinate and abscissa respectively. The readings taken by this means, though laborious, were accurate to within a few cycles per second and gave results that could be reproduced provided that the vibration remained constant throughout the period of the observation. The method was therefore applicable to a tool that was idling at constant speed, or to any tool in which the rotation speed was not dependent on the load applied to the cutting face. In practice it was found of little use with any machine under load because the small variations of speed from second to second made the 'peaks' on the meter impossible to follow. As a full set of readings might take up to 30 minutes the instrument could not be used to record the vibrations during a short operation such as striking a few blows with a pneumatic hammer, or a few seconds' grinding of an iron component against a wheel. A method giving instantaneous readings had to be sought.

The Acoustic Spectrometer

In the later stages an Acoustic Spectrometer, made by Messrs. Standard Telephones and Cables, Ltd., was used (fig. 2). In principle this is a high-speed waveform analyser, giving a visual indication in the form of a spectrum of the individual components of a complex wave. A block diagram of the electronic circuits is shown (fig. 3). The incoming signal from the pickup is first amplified, provision being made to vary the degree of amplification (the 'gain' control), and it is then applied to 27 band-pass filters of the wide type already described, arranged in parallel. The filters are designed to operate over an interval of 1/3rd of an octave each, and are constructed in such a way that if a signal is received in the middle of one pass range, the adjacent filters will pass only 20 per cent. of the actual amplitude. The frequency of the middle of the lowest band is 40 cycles per second, and that of the highest 16,000 per second. The output of each of the filter circuits is rectified and stored in condensers. In turn the output of the condensers is injected through a motor-driven rotating switch into a ring modulator and converted into alternating voltages of 3000 cycles per second. The output of the modulator is passed through a band pass filter to suppress harmonics and after further amplification and rectification is applied to the Y plates (vertical deflection) of the cathode ray tube, where a vertical band of fluorescence appears above a base line. The rotating switch makes contact with one of a series of constant potentials at the same time as it is applying the output of the storage condensers to the modulator. These fixed potentials are applied to the X plates (horizontal deflection) at the same time as the Y deflection, with the result that 27 separate spots or vertical lines appear on the screen (fig. 4). The height of each line is proportional to the vector sum of the amplitudes of the harmonic

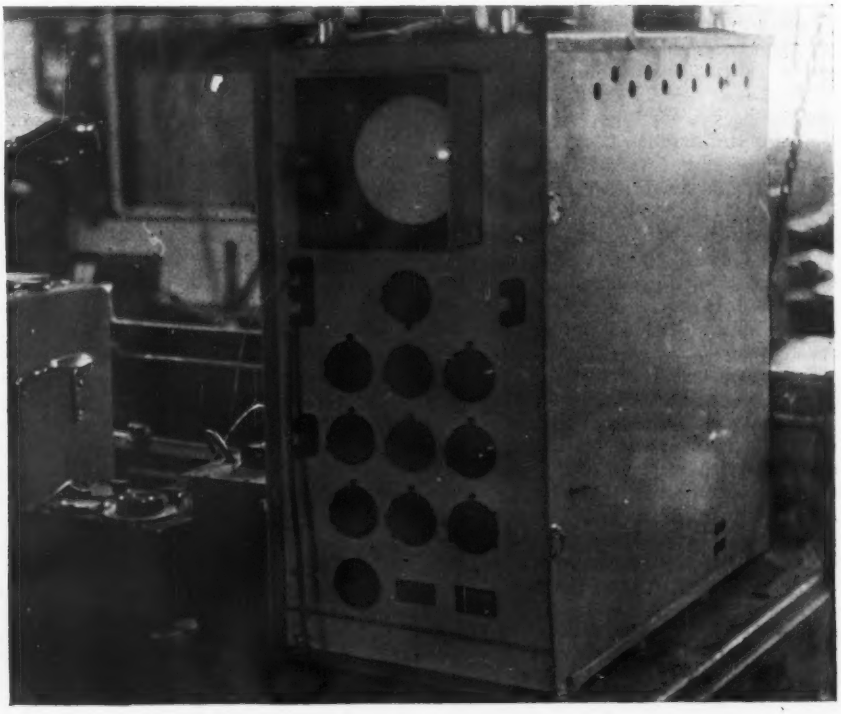

FIG. 2.—The Acoustic Spectrometer, with camera and hood removed.

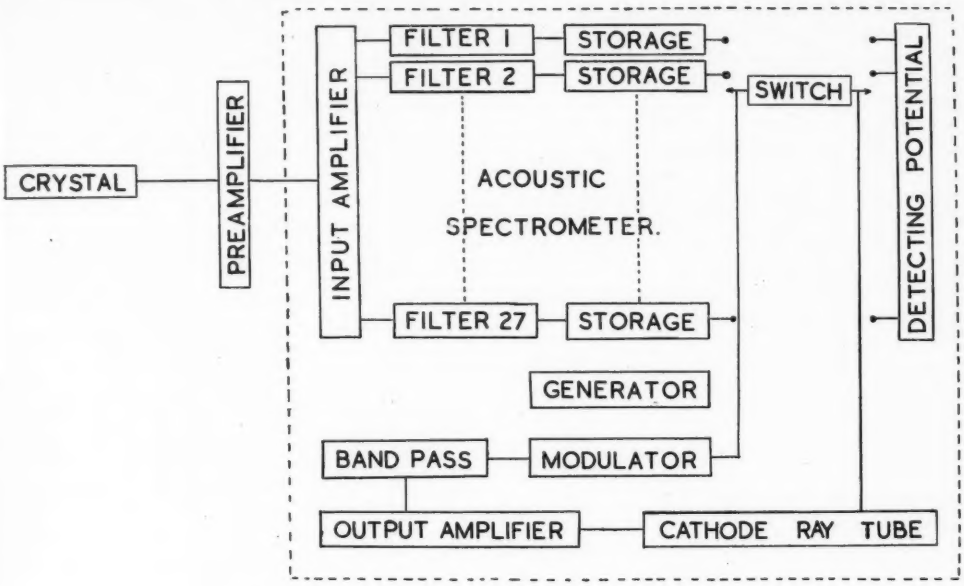

FIG. 3.—Block diagram of the electronic circuits in the Acoustic Spectrometer.

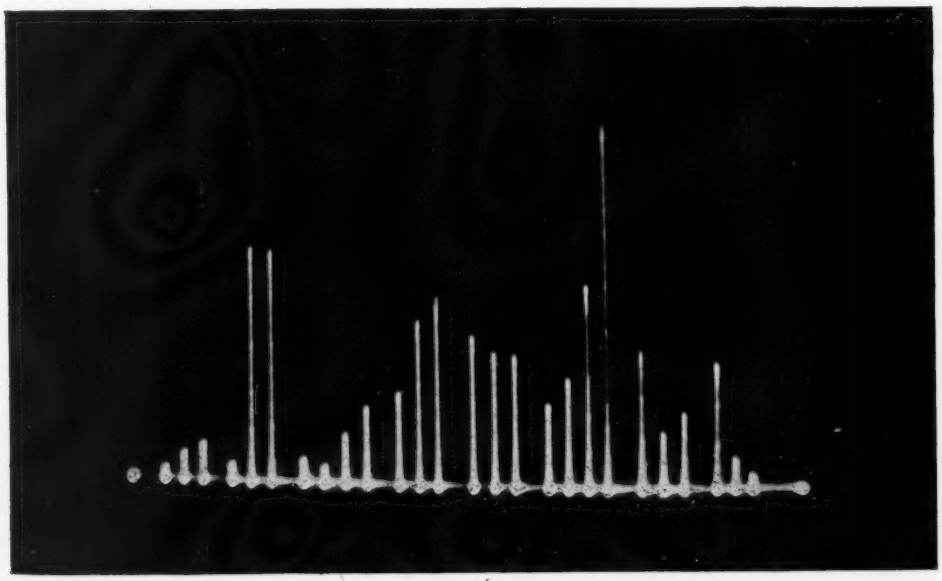

FIG. 4.—Photograph of the screen of the Acoustic Spectrometer taken while recording.

in the band it represents, and it can be measured by dividers, or alternatively the whole image can be photographed and measurements taken at leisure. The switch rotates at over 20 times per second, and due to this and the afterglow of the cathode-ray tube a continuous picture is obtained. The response of the filters is so arranged that, for an equal voltage of input, lines of equal length are drawn with an error of less than 10 per cent. A switch is incorporated whereby the charge and discharge times of the storage condensers can be altered. In the ' short ' position the charging time is approximately 20 milliseconds, and that of the discharge is about 100 milliseconds. In the ' long ' position these values are increased by a factor of 10.

To give a greater range of permissible input voltages, the crystal output was first applied to a separate amplifier whose characteristics were known to be linear in the range of frequencies with which this investigation is concerned. The gain stages of this amplifier were −20, 0, 20, 40, and 55 decibels. In addition to this the preamplifier had a much higher input impedance than that of the acoustic spectrometer, and was therefore well suited to receiving the crystal output.

The advantages of this instrument over the Harmonic Wave Analyser are that the whole of the vibration spectrum at any given moment can be seen at once and instantaneous records taken, so that many more readings are possible in a short time, and mean values can be assessed for any period in which much variation is taking place, by taking several records rapidly. The disadvantages are its bulk, the fact that it requires alternating mains current, and individual frequencies cannot be more accurately localized than within the filter-band limits already prescribed for the instrument. This makes it necessary to assume, until it is proved to the contrary, that the damaging frequencies will lie between certain narrowly defined limits but need not be individually determined with great accuracy; to assume the converse is to imply that all biological tissues are identical in behaviour. Small variations in the peaks due to the speed of the tool varying under load do not as a rule affect the response within a band, and this is advantageous.

Provision is made for the use of a cinematograph camera with the spectrograph, but it was found that the image on the screen varied very little from moment to moment when the tools were being worked, and not at all when they were idling. It was decided to use a 35-mm. miniature camera and to take individual exposures, one only for an idling tool, and three, for the calculation of mean values, for a tool under the variations of load. The camera was a Zeiss ' Contax ' with an f.2-Sonnar lens, mounted at 26·3 cm. from the cathode-ray tube screen in a detachable hooded mounting. To provide for the unusually short object-lens distance a tubular extension was made to fit between the detachable lens of the camera and its focal plane. The film used was Kodak ' Super XX ' Panchromatic, and it was developed under standard conditions to minimize any error from the shrinkage of film. The exposure required at full aperture was 1/10th second; more than 1/5th second caused halation, and less than 1/10th caused some of the image to be lost as the electron beam swept from one side of the screen to the other, unless the shutter happened by chance to be synchronous with one completed sweep. The lengths of the lines on the negative film were later measured on a simple microscope with a 3-inch objective and a micrometer scale in the eye-piece.

The mid-frequencies of the band-pass filters in the spectrometer, expressed in cycles per second, were as follows :

TABLE 1

1	40	10	320	19	2500
2	50	11	400	20	3200
3	64	12	500	21	4000
4	80	13	640	22	5000
5	100	14	800	23	6400
6	125	15	1000	24	8000
7	160	16	1250	25	10,000
8	200	17	1600	26	12,500
9	250	18	2000	27	16,000

Calibration of the Spectrometer

For the calibration of the preamplifier and the acoustic spectrometer the method used was to apply the output of a signal generator through a milli-voltmeter to the input of the preamplifier. The response of the spectrometer was measured on the screen with a steel rule. The amplifications provided by the various settings of the preamplifier were checked with standard input voltages and were found to be correct. The ' gain ' control of the spectrometer was in arbitrary divisions marked 0–130, and the instrument was calibrated as follows. The signal generator was tuned in succession to the centre of each band, and the voltage necessary to trace a line of 4 cm. on the screen was determined. In all cases it was found to be the same, within the limits given above. The signal generator was then tuned to one of three chosen frequencies, and, with the ' gain ' knob set in each of nine selected positions (130, 120, 110, . . . 50) readings were taken of the applied voltage necessary to produce a line measuring ½, 1, 2, . . . 8 cm. The mean of the readings for each of the three frequencies was calculated and plotted to give the family of curves shown in fig. 5. From this graph it is therefore possible to read off the millivolts input which corresponds to the length of the line appearing on the screen of the cathode-ray tube. A series of lines of standard length were produced and photographed. From this the ratio of the length of the image on the negative film to the length of the line on the screen was determined, and from this the alternative ordinate was inserted in fig. 5, which enables the length of line on the negative film to be converted directly into terms of input millivolts.

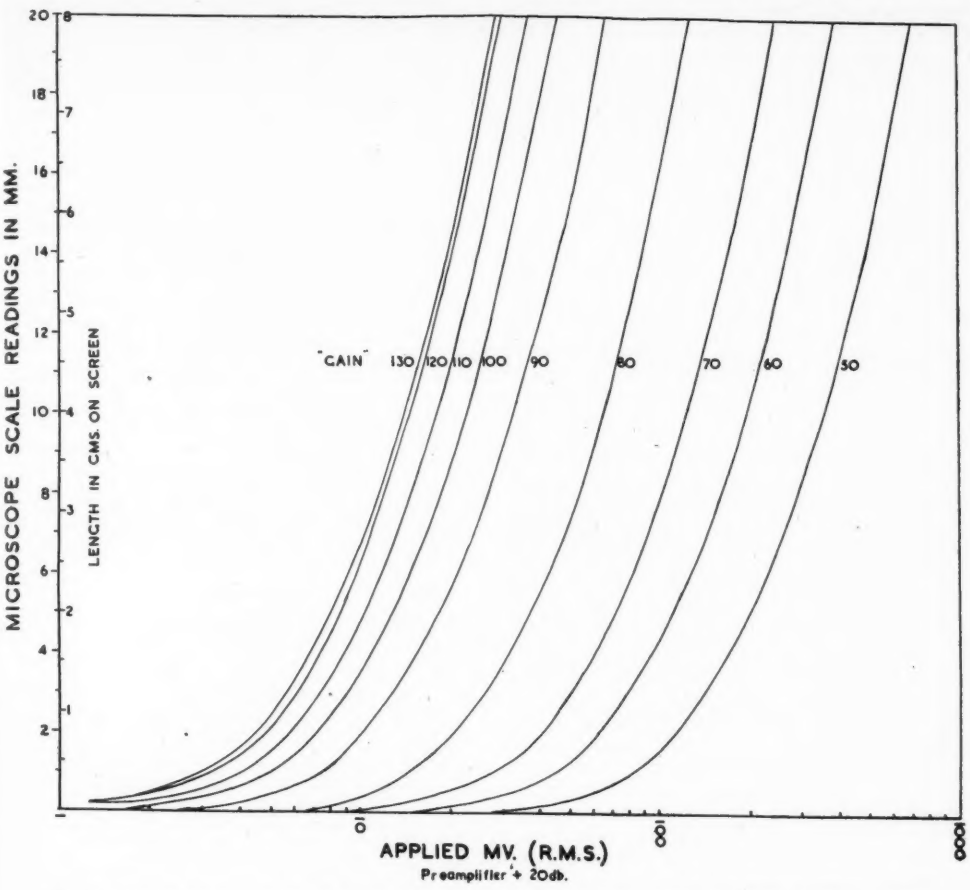
MICROSCOPE SCALE READINGS IN MM.
LENGTH IN CMS. ON SCREEN
"GAIN" 130 120 110 100 90 80 70 60 50
APPLIED MV. (R.M.S.)
Preamplifier + 20db.

FIG. 5.—Graph for conversion of length of the lines on the screen into millivolts of input voltage. (For explanation, see text.)

The voltage generated by a piezo crystal is given by

$$V = KAF^2$$

where V is the voltage generated
A is the amplitude of the vibration
F is the frequency of the vibration
K is a constant depending on the geometry and composition of the crystal.

The same voltage will therefore be generated by a crystal when subjected to a vibration of an amplitude of either 1 mm. at 100 cycles per second, or 10μ at 1000 cycles per second, or $0{\cdot}1\mu$ at 10,000 cycles per second.

It is necessary to bear this in mind always when viewing the whole spectrum on the cathode-ray tube screen, otherwise an erroneous impression will be gained because the higher frequencies will appear to have a much greater amplitude than is really the case after the correction has been applied. Similarly, a false impression would be obtained by measuring the total output of the crystal irrespective of the frequencies causing the generated voltage.

While it is not possible to deduce from the acoustic spectrometer records the amplitude corresponding to each individual frequency, a good approximation can be made by writing

$$V_b = K.F_b^2.A_b,$$

where the suffix b refers to the mid-frequency of the band-pass filter. If logarithms of both sides are taken the result is

$$\text{Log } V_b = \text{Log } KF_b^2 + \text{Log } A_b,$$

and it follows therefore that if V_b is plotted against A_b on log-by-log paper a series of parallel straight lines will be produced, all at 45 degrees to both axes, their position being determined by the value of F chosen. In this manner a second graph can be constructed enabling readings of V, the voltage generated as obtained from the first graph, to be converted directly into amplitudes, once the value of K is known. The makers of the crystals give data which leads to a value of

$$K = 1{\cdot}18 \times 10^{-5},$$

when V is expressed in millivolts and the amplitude

in microns. This value for K, which is based on data obtained at 1000 cycles per second, was checked by attaching the crystal to an electrically-maintained tuning fork suitably counterbalanced and running at a frequency of 100 cycles per second. The amplitude of the vibration was then determined microscopically as already described, and a value of K was calculated and found to be substantially in agreement with that obtained from the data supplied by the makers.

Specimen Result

Under investigation was a portable grinding tool which consisted of a metal hand-piece through which passed a flexible cable transmitting power from an electric motor suspended from the roof. The hand-piece was held by the operator in both his hands, and fastened in it was a mounted silicon carbide grinding stone of 1¾ inches diameter. The spindle of the stone was ¼ inch in diameter and was found to be 3½ thousandths of an inch out of true. The machine was rotating at 4050 r.p.m. as checked on a revolution counter, and the operator was grinding the smooth surface of a medium-sized hollow steel casting. The crystal mounting was strapped to the hand-piece directly under the palm of his left hand, which was placed in front. While he was working the response of the crystal was shown on the screen and produced a picture resembling that of fig. 4. The height of the highest lines was adjusted by using the 'gain' control to approximately 5 cm., and the position of this control noted. Three exposures were taken and the film processed. The lines reproduced on the negative film were measured and the mean value of the three taken. The lengths of the lines were converted into millivolts by the first graph (fig. 5), and thence into terms of amplitude in microns by using the second graph described above, and the results obtained were as follows :

TABLE 2

Frequency Band (cycles per second)	Amplitude (microns)	Frequency Band (cycles per second)	Amplitude (microns)
40	330	320	9·7
50	240	400	2·6
64	390	500	1·05
80	185	640	0·62
100	62	800	trace
125	94	1000	trace
160	47	1250	
200	37	and up-	nil.
250	24	wards	

By using such series of results comparisons are possible to show the differences between the vibrations reaching the right and the left hands, or the effect of interposing an absorbent rubber surface between the handle of the tool and the man's palm. These will be made the subject of a further communication.

Summary

The lesions which result from the absorption of vibrations, particularly Raynaud's phenomenon occuring in the hands, and the various types of work which may cause them, are briefly discussed. The type of vibrations set up, their analysis, and the usefulness of piezo-electric crystals for picking them up experimentally are considered. Two instruments for analysis of the response from the pickup crystals were used in experiments, the Harmonic Wave Analyser, and the Acoustic Spectrometer which superseded it. The design, calibration, methods of use, and the interpretation of the results from the latter are described, and a specimen series of readings is given.

The method has been applied to the measurement of the amplitude and frequencies of the harmonic components of complex vibration waves. No claim is made that the pathological processes leading to Raynaud's phenomenon and the bone and joint changes already known to be caused by vibrations depend upon these two physical quantities alone. It is probable that the total energy absorbed by the tissues has also to be considered.

Acknowledgments

We wish to acknowledge the help and encouragement of Dr. Donald Hunter, Director of the Department, and of Dr. Kenneth Perry. Our thanks are due to Professor D. T. Harris for help and advice, also to Mr. N. Fleming, Head of the Acoustics Section of the National Physical Laboratory, and Mr. B. H. Wallace, late of the Ford Motor Co. Without the very generous assistance of Dr. D. R. Thompson and members of the Vibration Department of the De Havilland Aircraft Co., Ltd., of Messrs. Marconi Instruments, Ltd., who lent the Harmonic Wave Analyser, and of the Controller of the Post Office Research Station, who lent the Acoustic Spectrometer, the work could not have been done.

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RAYNAUD'S PHENOMENON IN GRINDERS OF SMALL METAL CASTINGS

A CLINICAL AND EXPERIMENTAL STUDY

BY

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The clinical picture, now generally known as Raynaud's phenomenon, of intermittent pallor or cyanosis of the extremities precipitated by cold yet without evidence of blockage of the main peripheral vessels, can be brought about by a number of aetiological factors. There is no longer any doubt that vibration is one of these, even if the manner of its action is still uncertain. Pneumatic tools have for a long while been known to produce this syndrome in the hands after a latent period of some 2 to 5 years of working, and a number of clinical studies of the condition have been completed. It has been noted that right-handed men usually hold their pneumatic tool with the right hand on the handle at the back, and the left hand in front near the working end of the tool where it is subjected to the greater vibration. It is this left hand which is usually first and most extensively affected, even when protected with a glove. Moreover, though cold is in these cases a precipitating factor, it is not necessarily the cold exhaust air from the tool that is responsible, because this may be directed backwards towards the right hand, which may nevertheless be unaffected. A recent paper by Telford, McCann and MacCormack (1945) again suggests that it is the vibrations that are primarily responsible, for they describe the phenomenon occurring after the use of a small portable scurfing or grinding tool driven by a flexible cable drive from a suspended electric motor. The grip employed on the hand-piece of this tool is usually an overhand grip with the left hand and an underhand with the right, the left being in front. When using the tool they describe, the impression is gained that the left hand is subjected to rather more vibration than the right, and more pressure is needed with this hand to hold the rotating cutting-head against the metal to be worked upon. The left hand once more is the first affected, after an average delay of two years and three months from starting to use the tool, regardless of the age of the worker.

A number of typical examples of the Raynaud phenomenon have been observed in the last two years in the grinding shop of an iron and steel foundry in the home counties. In these cases the vibration is transmitted from the grinding surface to the hands via the articles being ground, and not because of the use of any portable tool which itself vibrates. It is the purpose of this paper to describe the clinical findings in a number of such cases and the results of an analysis of the type of vibration encountered.

The Method of Grinding

The foundry produces a number of iron components of various shapes and sizes which constitute the angle, T-piece, and joining connections for systems of iron piping. The rough castings come to the grinding shop to have the runners and other excess metal removed from the lines where the two halves of the casting mould were joined. This grinding takes place prior to machining and galvanizing. Some of the components are to fit pipes of some 4 inches internal diameter, but most of them are smaller, of $\frac{3}{4}$ inch internal diameter or less. They are ground against large plastic-bonded carborundum wheels, most of which are belt-driven; a few are on electrically driven spindles. The heavier work is mainly done by men, but for some of the lighter work and most of the final grinding on the electrically operated wheels women have of late been employed. All the wheels are of comparable diameter, and different sizes of pulleys allow of three speeds of drive to enable an increase in the revolutions to be made as the wheel becomes worn, to maintain the surface speed at the optimum figure. The thickness of the wheel is usually either $2\frac{3}{4}$ inches for the larger pieces and the outer surfaces, or of $\frac{1}{4}$ to $\frac{3}{8}$ inch for the less accessible internal angles. The speeds for a typical one of the thicker wheels vary between 730, 912, and 950 r.p.m., the alterations being made as the wheel wears down to diameters of 30, $24\frac{1}{2}$ and 20 inches respectively. This means that the velocity of the grinding surface varies between the limits of 4970 and 5840 surface feet per minute. For a narrow wheel the revolutions are initially 1950 per minute to correspond to a diameter of 16 inches, and finally 3050 per minute to correspond with 8 inches diameter. The limits are in this case 6390 to 8180 surface feet per minute. The electrically operated wheels are of $1\frac{1}{2}$ and 3 inches thickness and work respectively at 7310 to 9180, and 2590 to 6650, surface feet per minute.

The workman stands at his wheel with a tray of rough castings within easy reach to his left and an empty tray to his right into which he tosses the

finished components. As the work is piece-work he will achieve considerable manual dexterity in handling them, and can grind one piece, sides and ends, in some two or three seconds. He grips it firmly between thumb and index finger of each hand; he may use the middle finger also in handling the larger pieces, but the remaining fingers usually rest lightly on an adjustable steel bench or anvil (fig. 1)

FIG. 1.—General view of belt-driven grinding equipment, showing adjustable steel bench, various tongs, and a tray of ground components.

close to the wheel, and are seldom in direct contact with the metal being ground. The main downward pressure is taken by this fixed bench, but the horizontal pressure for pushing the casting against the wheel is provided by his fingers, and in particular the terminal portion of the thumb. The work may only be in contact with the wheel for less than a second at a time, but during this time an intense vibration is set up. The manoeuvres are repeated many hundreds of times in an hour, and the men have averaged a 50-hour week. A man may elect to wear gloves while grinding, but this is unusual. Alternatively he may bind his thumbs and index fingers with strips of cloth, but he does so with a view to lessening the risk of trauma to his fingers or burns from the heat of the grinding process, and not to reduce the vibrations.

Some of the men have worked at a broad wheel or a narrow one for the whole of their employment, but several have changed from time to time. Each man is liable to change the type of component he grinds almost from hour to hour. In handling the smaller ones he may choose to use a pair of rough metal tongs with which to grip the wall of the hollow casting; the tongs are held in the palm of the right hand. Most of them prefer to use these tongs as in this way greater control is obtained, and again the risk of trauma and burning of the fingers is lessened. To press components on to the broader wheels more effort is needed, and the man is provided with a wooden 'crutch', of which one end fits into a slot in the back of the metal bench and the other rests against his upper abdomen, being steadied there with his left hand from time to time

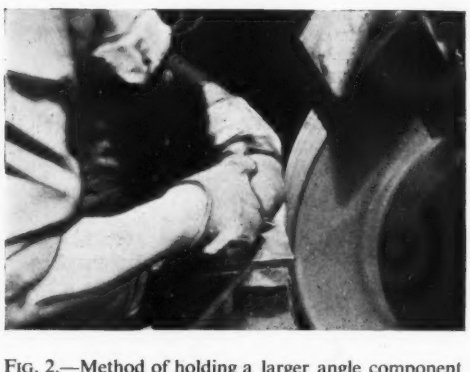

FIG. 2.—Method of holding a larger angle component in the hands for grinding its outer surface.

(fig. 3). By leaning forwards on this crutch he may use his weight to press the casting forwards, and he has one hand relatively free. Use of the crutch clearly lessens the quantity of vibration transmitted to his fingers. From time to time while working a man finds the surface of his wheel has become too rough, and he dresses it for a few seconds by grinding a special serrated tool of hard steel.

The grinding shop is airy and well ventilated, but noisy. It is usually warm, and the men are naturally handling warm articles all day since the grinding process makes them so. Goggles for protection against flying fragments are invariably worn.

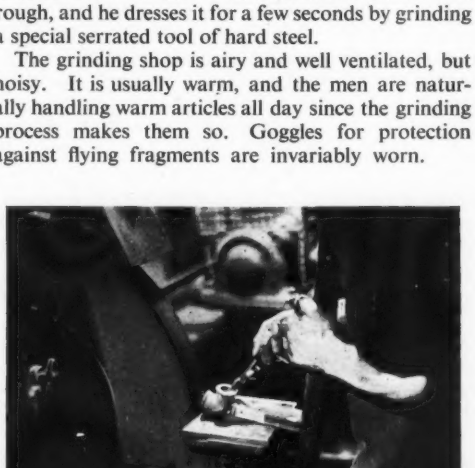

FIG. 3.—Showing the use of the tongs and the 'crutch'.

Clinical Findings

No intentional selection of cases was made. All members of the grinding shop who would consent were interviewed, whether or not they were known to exhibit Raynaud's phenomenon. Only one or two refused. Workers who had worked at grinding previously but who had since changed their occupation within the same works were also included. This change had in one or two instances been made at the medical officer's recommendation, as a result of complaints of 'dead fingers', but in other cases the events were unconnected. It was noticed throughout the investigation that the workers were in general very little concerned with their lesions in spite of the fact that several of them suffered some inconvenience. In most cases they had not bothered to complain to the doctor previously, but they had named the condition 'grinders cramp' amongst themselves and attributed it to the tight gripping of castings.

A full occupational history was taken, also a routine medical history in which particular regard was paid to previous vascular disturbances, or any familial incidence of these. Clinical examinations followed, and each individual was subjected to a water immersion test, being made to sit for fifteen minutes with the arms below the level of the heart, immersed to above the elbows in water which was kept as nearly as possible to 15° C., after which the distribution of pallor or cyanosis in the fingers was noted.

In all 37 workers were seen, 29 men and 8 women, and of the 29 men, there were 27 who showed the phenomenon and 2 who did not, while of the 8 women, 5 showed it and 3 did not. These figures result from considering the claims of the individuals and not from the outcome of the immersion test. Of the two negative male cases, one was aged 21 and had worked in the grinding shop for 1 year, while the other was 51 years old, had worked in this grinding shop for 6 years, and had 13½ years of similar work to his credit. The negative cases amongst the women were aged 35, 21, and 42, and had worked respectively for 6 months, 1 year, and 3 years and 9 months on the smaller of the electrically driven wheels. Of the positive male cases the first gave a self-contradictory history, and it was thought advisable to exclude his figures from the analysis.

All the workers were right-handed except for one man and one woman. Considering the positive cases, the age limits for the men were 29 to 50 with a mean of 37, while the age limits for the women were 24 to 45, with a mean of 36 years. The average duration of working for all of the positive cases was 5 years and 3 months at the time of the questioning; the limits were wide, lying between 1 and 12 years. Assessing the time that elapsed between starting this work and the onset of symptoms the average was 1 year and 9 months, though the limits were 0 to 7 years. In this connection it is as well to note, first, that a man may find much more difficulty in stating how long his symptoms have been present than he will in remembering the duration of his employment; the last figures therefore tend to be inaccurate. Secondly, two of the men reported that the symptoms first came on before they ever started work at grinding, at 2 and 2½ years before respectively. The reason for this is obscure and may perhaps point to a general increase of vascular disturbances of this type amongst the population during recent years, but the high incidence amongst the 37 people comprising this present series is clearly too much to be explained by this means. In neither of these last two unusual cases was there evidence that previously the men had used vibrating tools or had done grinding work.

On direct questioning two workers gave a previous history of having suffered from deadness of the fingers earlier in life, but in neither case did the severity or distribution appear to correspond with that after grinding work had begun. None would admit to any similar disease in his family; in a series of 37 that is perhaps questionable, but is probably due to faulty observation. Three of the men had previously done work with grinding wheels but on different castings and in different circumstances; the figures in their cases are therefore included, though the possibility that this previous work influenced their present condition is not denied. One of them was remarkable for being in the negative group with a total of 19½ years of service at grinding in various forms. The second had had 10 years experience of grinding and fettling previously, yet had only developed the syndrome 2 years after starting work in this grinding shop. The third had had a year of grinding nearly 10 years previously, but here again the onset of symptoms had been 2 years after beginning his present job.

In 2 cases the right hand had been affected first, in 11 the left; in the 18 others the onset was in both hands simultaneously. One left-handed worker had her left hand affected first, the other had both his at once. Of those originally affected in one hand only, six said that eventually both were involved, so that ultimately 23 out of the 31 admitted to bilateral lesions, with a tendency for the left to be involved first. All were found to be able to indicate

TABLE I

DISTRIBUTION OF RAYNAUD'S PHENOMENON IN 31 CASES

Extent	Right hand					Left hand				
	Th.	II	III	IV	V	Th.	II	III	IV	V
Tip to distal interphalangeal joint	–	19	18	16	13	–	22	27	23	18
Tip to proximal interphalangeal joint	6	17	15	14	10	9	20	24	21	14
Tip to metacarpophalangeal joint	1	6	5	4	5	2	10	8	5	5

with fair certainty how far down the fingers the lesion usually spread, for, as is usual in this condition, in any affected fingers the tips were invariably involved and the pallor would extend proximally. The workers were asked to estimate the extent of their lesions in terms of joints of the fingers, and the results are given in Table 1, which is based on statements and not on the immersion test.

Cyanosis in place of pallor or following upon it was admitted by 8 patients, but denied in 23. In the 8 cases there was insufficient evidence to show whether cyanosis can be considered a sign of later onset than pallor, or whether cyanosis can be considered to indicate a lesion of particular severity. The distribution in terms of number of digits affected was so wide that it did not suggest this either. In all cases it was confirmed that cold conditions precipitated an attack, and in no case

TABLE 2
SUMMARY OF THE CLINICAL FINDINGS IN 37 GRINDERS

Case No.	Sex	Age	R. or L. handed	Years employed at this type of grinding	Onset of Raynaud's from start of this occupation (years)	Type of wheel	Hand of onset	Present distribution		Cyanosis claimed	Onset at work claimed	Some disability
								Right hand	Left hand			
1	M	17	R	5/12	1/12	Large	R	2‴3‴	—	−	+	−
2	M	32	R	6	1	Small	R	2″ 3″4″	—	−	−	+
3	M	39	R	2 2/12	1 7/12	Large	L	—	3″4″	−	+	−
4	M	41	R	2	0	Large	R & L	2′ 3′ 4′	2′ 3′ 4′	−	+	+
5	M	42	R	5 8/12	2 8/12	Small	R & L	1′ 2″3″4″5′	1′ 2″ 3″4″5′	+	−	−
6	M	33	R	5 9/12	1	Large	R & L	1′ 2″3″4″5′	1′ 2″ 3″4″5′	−	−	−
7	M	50	R	6	2	Large	R & L	2″3″4″5″	2″ 3″4″5″	−	+	+
8	M	32	R	6	?	Small	L	—	2‴3″4″5′	+	−	+
9	M	41	R	2	?	Both	L	—	1′ 2‴3‴	+	+	−
10	M	38	R	1 6/12	11/12	Small	R & L	4′	1′ 2″	+	−	−
11	M	36	R	1	4/12	Small	R & L	2‴	1″2‴	−	−	−
12	M	33	R	7	6/12	Large	R & L	2‴3‴4‴5‴	2‴3‴4‴5‴	+	+	+
13	M	29	R	2	0	Large	R & L	5′	3″4″5″	+	−	−
14	M	21	R	1	—	Both	—	—	—	−	−	−
15	M	51	R	6	—	Both	—	—	—	−	−	−
16	M	34	R	6 6/12	2	Small	L	—	2′ 3′	−	+	−
17	M	42	R	6 6/12	2	Large	R & L	3″ 4″ 5″	3″ 4″ 5″	+	+	+
18	M	43	R	11 6/12	2	Small	R & L	3′ 4″ 5‴	3′ 4″ 5‴	+	−	−
19	M	31	R	5 6/12	4 6/12	Large	L	3″	2″ 3″ 4″ 5″	−	+	+
20	M	38	R	12	7	Large	R	2‴3‴4‴5‴	2‴3‴4‴5‴	−	+	+
21	M	38	L	5 9/12	1 3/12	Large	R & L	2″ 3″ 4″	2″ 3″ 4″	−	−	−
22	M	34	R	11	1	Large	R & L	1″2″ 3″ 4″ 5″	1″2″ 3″ 4″ 5″	−	−	+
23	M	30	R	8 6/12	1 6/12	Small	L	2″ 3″ 4″ 5″	2″ 3″ 4″ 5″	−	−	+
24	M	40	R	11 6/12	6/12	Large	R & L	2‴3‴4‴5‴	2‴3‴4‴5‴	−	+	+
25	M	35	R	5	1	Both	R & L	1′ 2‴3‴4‴5‴	1′ 2‴3‴4‴5‴	−	+	+
26	M	39	R	1	6/12	Large	L	2′ 3′	3″ 4″ 5″	−	−	−
27	M	41	R	4	1	Small	L	—	2‴3‴4‴	−	+	−
28	M	34	R	2	1	Large	R & L	2″	3″	−	+	+
29	M	41	R	8	2	Small	L	—	3″4″5″	−	+	−
30	F	40	R	3	1 6/12	Small	R & L	2‴3‴	2‴3‴	−	+	−
31	F	39	R	3 9/12	1 9/12	Small	R & L	1′ 2″ 3″4″5″	1′ 2″ 3″4″5″	−	+	+
32	F	24	R	2 6/12	1 10/12	Both	L	—	3″4′	−	+	+
33	F	32	R	2 6/12	1	Small	R & L	2″	2″	−	−	−
34	F	45	L	4	1	Both	L	1′ 2″	1′ 2‴3‴4″ 5′	−	+	−
35	F	35	R	6/12	—	Small	—	—	—	−	−	−
36	F	21	R	1	—	Small	—	—	—	−	−	−
37	F	42	R	3 9/12	—	Small	—	—	—	−	−	−

(*Note* 1.—For convenience in describing the present distribution the convention is used that the number of strokes following the number of the digit indicates the anatomical spread of the lesion, according to Table 1.
2.—Case No. 1 excluded from final analysis—see text.)

was it admitted that attacks came on under stress of emotion. One man said that he had pain during the attacks, another complained of pain if he continued to work during an attack, while two others talked of a dull ache. There were a number who described a painful tingling sensation as the blood returned to the affected fingers. Fifteen patients said that attacks could come on while they were working; 13 denied that this ever happened though they were often affected in the early morning on rising; 3 others had attacks at the start of a day's work but thereafter were not troubled. The duration of individual attacks appeared to vary between 10 and 180 minutes, but this was at best given in round figures, many saying that the attacks lasted until their hands became warm again. There was little evidence to suggest either that a widespread lesion tended to have a long duration, or the converse.

The disability caused to the workers was very difficult to assess, though generally it was minimized, and anxiety as to the future was very rarely seen. Some inconvenience was alleged by 15 people, 11 of these being amongst those whose attacks came on while working; 4 claimed some disability even though attacks were not apparent at work. There was no evidence of the loss of whole days of work from this cause, but in a few cases a man might lose an hour or two through having to wait while his hands warmed up, a point of some significance in piece-work. There were 12 who had for various reasons given up grinding; three of them denied any improvement as a result though they had stopped for 2 months, 1 year, and 5½ years respectively. The remainder claimed some degree of improvement, and in one case attacks had ceased 1 year after stopping the work. In most cases it was not possible to define a man's evidence for claiming he was improved. It seems likely that if questioned at the end of the summer more people will admit improvement than at the end of the winter.

There were few abnormal physical signs to see. Two men had a mild degree of hypertension with a systolic blood pressure of 170 mm. Hg. One was aged 33, and had extensive bilateral involvement of his fingers which came on after a year of this work, while the other was aged 38 and had slight involvement coming on 11 months after starting. In two cases there were punctate and linear burn scars extending up both forearms from the showers of burning sparks that are given off in grinding. In one or two cases there were unusually placed callosities, but there was no evidence of trophic or neurological changes in the fingers.

The water immersion test yielded little additional information, but had to be done on a warm day under conditions that were difficult to control. In 21 of the cases pallor or cyanosis was produced, and in 10 others who alleged they had the disease there was nothing abnormal to see, though 2 of these had claimed extensive lesions. In most cases the water test produced signs that were less extensive than those claimed, and the impression gained was that in the conditions of a factory first aid station there are too many variable factors to make the test of much value, and that it ought not to be used as a diagnostic test of a man's claims in cases of dispute.

The cases were found to be divisible into three groups, those where the larger grinding wheels had been used throughout, those where the smaller, and those where there had been a change from one to the other. If the extent of the lesion is expressed in terms of the number of portions of finger involved, according to Table 1, and if extent of lesion can be assumed to give some measure of severity of a particular case, some correlation might appear between severity of the lesion and the type of grinding done on the large or the small wheels, provided that in the two groups there is no great discrepancy between ages or periods of service. Taking into consideration both negative and positive cases, there are 16 who used the small wheels, 14 who used the large, and 6 who had used both at various times. The average age of those who used the small was 36·1, and of those who used the large 37·2. The scatter is shown in the following table.

TABLE 3

Age group	Large wheel	Small wheel
Under 30 ..	1	1
30–34 ..	5	5
35–39 ..	4	4
40–44 ..	3	6
45 and over ..	1	0
Total	14	16

The two groups are therefore reasonably comparable as regards age. Further analysis then shows :

TABLE 4

Grinders using:	No. of obs.	Length of employment		Areas affected	
		Mean	Standard deviation	Mean	Standard deviation
Small wheel ..	16	4·58	3·00	7·69	5·72
Large wheel ..	14	5·73	3·58	13·36	7·13

This indicates, in spite of the fact that the numbers are small and the standard deviations large, that there is a difference between the means of the portions of digits affected (i.e. the 'severity' of the lesions) of 13·36 − 7·69 = 5·67. There is a small difference between the mean length of employment

TABLE 5

Duration of employment in years	Mean Values								
	Small wheel			Large wheel			Both wheels		
	No.	Age	'Severity'	No.	Age	'Severity'	No.	Age	'Severity'
Under 3	5	32·4	3·2	5	36·4	5·8	3	28·7	3·3
3–5	5	40·8	11·0	3	34·0	12·7	2	40·0	19·5
6 and over	6	35·3	8·7	6	39·5	20·0	1	51·0	0

in the two groups, but scarcely enough to account for this difference in 'severity'. The difference of 5·67 between the means is more than would have been likely to have arisen by chance (P lying between 0·05 and 0·02 with the 't' test). This may indicate that the kind of work done with the larger wheels causes a more extensive distribution of the lesion than the kind done with the smaller.

It was found on questioning the workers that some would admit that the lesions had got progressively more extensive, some denied this, but a high proportion were uncertain. An attempt was therefore made to settle the point on statistical evidence. Taking, in Table 5, three groups according to duration of employment at this form of grinding, and considering the means of the ages and of the 'severity' expressed as before in terms of the number of portions of digits affected, the analysis appears as above.

This suggests that there is some evidence in the case of grinding operations on the large wheels that the severity of the lesion depends on the duration of a man's employment. The possibility of age affecting this issue cannot be quite excluded. Taking together the employees on small and large wheels, there is a correlation coefficient between the individual duration of employment and the index of severity of +0·65; this is a significant degree of association.

Analysis of the Vibrations

Using the method of two of the authors (J. N. A. and H. A. D.) described elsewhere in this journal, an analysis was attempted of the type of vibrations set up during grinding work. The method involves the use of a piezo-electric crystal pickup and an acoustic spectrometer, and its rapidity of recording was particularly useful here because each individual grinding process is of very short duration, in some cases even less than one second. It is to be expected that during each action of grinding the total vibration would build up to a peak and then be reduced again as the worker increases and decreases the pressure, and as the wheel takes up the load on a rough surface and polishes it smooth. A composite picture of the vibration in relation to the duration of the whole grinding 'stroke' could be obtained by using a cinematograph camera technique, but this has not yet been attempted. Instead, single records were taken when the vibration had reached its maximum as far as could be ascertained. During the grinding of one component it may be turned in all directions, and applied to the wheel for varying periods and at different pressures. The records show a wide variability which is in keeping with this, and it must be emphasized that comparisons can only be made when one factor alone has been allowed to alter. Any comparison of the differences in the vibration obtained from grinding different shapes and sizes of component, or from different sizes of wheel for different parts of the same component, are of limited value only.

In the first case a narrow wheel was being used to grind the angles of a small T-junction piece held in the fingers. The crystal was placed under the left thumb on the upper surface of the casting, facing downwards on to the bench. Normally such a piece is held with tongs in the right hand, and for comparison the crystal was next placed in the palm of the worker's right hand and held firmly against one of the handles of the tongs, all other conditions being kept unchanged. The results appear in Table 6.

This shows that there is a very similar vibration reaching the two hands despite the use of the tongs

TABLE 6

Frequency (cycles per sec.)	Amplitude (microns)	
	Crystal on metal, under L. thumb	Crystal on tongs in R. palm
40	—	630
50	—	—
64	350	600
80	155	240
100	122	195
125	105	100
160	64	69
200	33	35
250	28	31
320	28	22
400	20	16
500	16	10
640	8	9
800	4	4
1000	2	2
1250	2	2
1600	1	1
2000–10000	trace	trace

in the right. The major frequency is differently placed in the two, and the amplitudes are large. The dimensions of this component were some 2 inches by 1 inch, and ¾ inch internal diameter.

The following figures (Table 7) were obtained by placing the crystal at one point on an angle component of larger size, some 4 inches internal diameter and 8 inches overall length. First one end was ground, then the piece was placed flat on the bench and the relatively smooth runner removed from the whole length of the outer surface, and finally the other open end was ground, and this was rough. Though the crystal was fixed in one position on the casting and always under the palm of the left hand, its plane in relation to the grinding wheel surface was altered three times as the workman went through the three manoeuvres.

TABLE 7

Frequency (cycles per second)	Amplitude (microns)		
	End	External runner	End
40	—	—	—
50	—	—	—
64	—	—	—
80	—	—	220
100	—	—	162
125	—	—	96
160	—	—	67
200	—	—	43
250	—	—	31
320	—	—	19
400	—	—	16
500	—	—	8·2
640	—	—	5·9
800	—	—	2·5
1000	—	—	1·4
1250	—	—	1·1
1600	—	0·95	1·4
2000	—	0·62	0·48
2050	—	0·23	0·2
3200	0·16	0·12	0·19
4000–10,000	trace	trace	trace

This series illustrates the variability that is likely to be met in a single set of operations, and that under certain circumstances of grinding a large component the total vibration may be of small quantity, lying at the high-frequency end of the spectrum, having small individual amplitudes, and consisting in fact of the natural 'ring' of the metal only. The conditions are most likely to be fulfilled when the casting is lying in a stable position flat on the bench.

A final set of readings to be considered relates to the grinding of the outer surface runner on a medium-sized casting, again hollow as all these components are, and some 2 inches in internal diameter, 4 inches in overall length, and right-angled in shape. In the first instance the pickup was placed on the inner surface of the angle such that it lay vertically in space with its surface in the same plane as the surface of the wheel itself. In the second instance the pickup was flat upon the top surface of the casting, its plane being that of the surface of the steel bench (Table 8).

TABLE 8

Frequency (cycles per second)	Amplitude (microns)	
	Crystal pickup vertical	Crystal pickup flat
40	—	255
50	—	—
64	—	100
80	—	80
100	—	51
125	—	43
160	—	53
200	—	32
250	13·5	26·5
320	9·6	14
400	6·1	9·6
500	—	6·4
640	1·95	3·7
800	1·75	2·0
1000	1·5	2·1
1250	1·05	1·9
1600	0·51	0·57
2000–3200	trace	trace
4000–10,000	trace	nil

These figures, besides indicating the wide variability to be expected, show that in any one operation of grinding the vibration takes place in at least two dimensions, for the crystal pickup only responds effectively to vibration in a plane at right angles to its flat surface. In the case just quoted the conditions in the two halves of the experiment were kept identical, only the plane of the pickup being changed. When in a vertical position the total vibration is small, nothing is encountered below 250 cycles, and even the greatest amplitude is small. When in the horizontal position the frequencies extend throughout the spectrum, and at the lower frequencies the amplitudes are large. This indicates that the vibration taking place in a direction at right angles to the surface of the wheel is slight, but that in a vertical direction, i.e. tangential to the wheel surface, it is large. Little assessment of the quantity reaching the fingers can be made, because they are wrapped round such a casting in all directions and can therefore absorb vibration in various planes at once.

In all these experiments means have been taken of either three or six readings, but in any one experiment the variation from moment to moment was surprisingly small. Further records were taken to consider other aspects of the grinding, but they serve to emphasize the variability of the vibrations which is in keeping with the many variable experimental conditions met. A series was contemplated to compare the effects of grinding a given component on wheels of different sizes, but this would have had little practical application, as it is rarely seen that one particular type is ground on both the

large and the small wheels. In general terms little difference between the effects of the various types of wheel could be detected, though detailed comparisons are inadmissable for the reasons already given.

Discussion

An assessment of the degree of disability in this condition is notoriously difficult. In the paper by Telford, McCann and MacCormack (1945), the cases exhibited an identical syndrome from a different type of work, but were not a true cross-section of all the workers on the job, for they were a selected group made up of those presenting with definite symptoms and therefore above the average in severity. The disability was described there as 'severe', and the uselessness of the hand during the attacks was emphasized. But it is only the cutaneous sensibility that is lost, not the muscular power. Grinding requires muscular power but not much delicacy of touch. Therefore, what is disability for a former textile worker about to return to his old job may not constitute disability for a grinder. Similarly, an outdoor worker may have more cause for complaint than an indoor one, because he is liable to more frequent attacks from the cold. In the present series there has been nothing to suggest that the more serious lesions, such as trophic skin changes or even gangrene, are to be expected.

It is worth noting that in our series the men were mostly contented and in many cases did the work from choice; they had seldom been directed from other occupations. This may have considerable bearing on their lack of concern for their symptoms. In other parts of the country where Raynaud's phenomenon occurs also from working conditions, more disability is claimed where there has been unwelcome direction of labour, or where men have suddenly been declared redundant after having had a lucrative job.

Cases have been coming to the fore in the last two or three years even though grinding processes such as this have been in use for many years. Whether this is a function of a tendency in the general population, or of the specific effect of increased pressure of work and longer hours combined with more medical supervision in factories, or whether it is from all these causes coupled by some such factor as dietary deficiencies in wartime, it is not yet possible to say.

At present there appears to be no possible prophylactic measure that can be applied. If all the work could be done by machinery the problem would be solved, but the skill and speed required and the complexity of the manipulations make this scarcely possible. Hand grinding will always produce vibration, and the best that can be hoped for is some method in which the man touches the metal as little as possible, or some suitable absorbent material to cover his fingers without hindering the work unduly.

Summary

Thirty-seven workers from a grinding shop were examined, and thirty-two had Raynaud's phenomenon in cold weather. The type of work and the grinding equipment is described. Clinical findings suggested that the lesions were mostly bilateral, came on at an average of 21 months from the time of starting the work, and caused relatively little disability; the exact distribution of the lesions was noted. There is some evidence to suggest that the processes of grinding with the larger wheels cause a more widespead lesion than others; also that the disturbance becomes more extensive the longer that work with a large wheel goes on. Experimental work on the quality of the vibrations set up shows the order of the frequencies and amplitudes concerned, the considerable variability, and the fact that vibration is taking place in several directions at once.

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A REPORT OF THREE CASES OF ACCIDENTAL POISONING BY SODIUM TELLURITE

BY

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The rarity of poisoning by the salts of tellurium warrants the reporting of the present cases, together with a note upon the experimental findings.

Case Reports

Case 1. Male subject aged 40. Transferred from another hospital on 20/10/44 with a six months' history of left renal pain associated with frequency of micturition. Investigation in the previous hospital had revealed evidence of cystitis and also of enlargement of the left kidney after intravenous pyelography. On admission the urine contained a trace of albumin, but otherwise there were no abnormal physical findings. The development of a mild coryzal attack after admission delayed further investigations. On 30/10/44 after premedication with omnopon gr. $\frac{1}{3}$ and scopolamine gr. 1/150, 0.65 gm. of sodium pentothal was administered intravenously at 9.30 a.m. and a cystiscope passed. Erosion of the bladder adjoining the left ureteric orifice was observed. Difficulty was experienced in passing a ureteric catheter into the left ureter and on completion of the operation 15 c.c. of ' sodium iodide ' solution were injected. The patient left the theatre at approximately 10 a.m. and was fully conscious. A peculiar garlic smell in the proximity of the patient was observed at 11.30 a.m. At 12.45 p.m. the patient complained of severe discomfort in the left loin and was ordered morphine gr. $\frac{1}{4}$. At 1 p.m. the patient vomited. 2 p.m. the patient was deeply cyanosed, dyspnoeic, complained of nausea and vomited. 2.20 p.m. patient died. Autopsy performed 20 hours after death.

Case 2. Male subject aged 21. Admitted to hospital with burns of foot and whilst under treatment developed frequency of micturition and pain in the loins. The urine contained numerous pus cells and on culture yielded a profuse growth of bacillus coli. The patient was ordered a course of sulphathiazol (total 18 gm.) and the urine became sterile, but the infection reappeared five days later. 21/9/44, a course of calcium mandelate and ammonium chloride was prescribed without effect so the patient was investigated further. Cystoscopic examination revealed a mild degree of cystitis; intravenous pyelography suggested defective secretion of the left kidney. On 28/10/44 after preliminary medication with omnopon gr. $\frac{1}{3}$ and scopolamine gr. 1/150, 0.65 gm. of sodium pentothal was administered, a cystoscope passed and a ureteric catheter introduced into the left ureter. On injecting the " sodium iodide " solution the catheter appeared to be blocked and was withdrawn. On return to the ward the patient felt well and made no complaint. 2/11/44 the sister in charge of the ward detected a pungent garlic odour emanating from the patient who complained of a nasty taste in the mouth. In view of Case 1 fluids were pushed, intravenous glucose administered but the patient himself made no complaints. The only abnormal physical finding was a slight degree of cyanosis. 3/11/44 patient vomited a small amount of fluid on three occasions. 5/11/44 patient was well and had no further vomiting; urine output was normal and no abnormal physical findings were detected. Spectroscopic examination of the blood showed bands of methaemoglobin. Red blood cells 5,030,000, haemoglobin 100 per cent. (Sahli) white blood cells total count 8000, polymorphs 73 per cent., small lymphocytes 22 per cent., large lymphocytes 1 per cent., monocytes 4 per cent. The urine contained a trace of albumin. The centrifuged deposit contained granular and hyaline casts. Culture yielded a profuse growth of bacillus coli. The patient made a complete recovery, no further untoward symptoms developed and the garlic foetor of the breath had disappeared on 10/11/44.

Case 3. Male aged 31. Transferred to hospital on 22/12/44 for the investigation of left renal colic of three months duration. Only other complaint was a mild degree of backache after exertion since 1941. Physical examination revealed nothing abnormal, BP 130/85. Urine output was normal, and there were no abnormal constituents. Straight x-ray examination of the renal tract and intravenous pyelography revealed no abnormality. In view of severity of symptoms cystoscopy and retrograde pyelography was advised. 30/12/44 Cystoscopic examination was made after preliminary medication with omnopon gr. $\frac{1}{3}$ and scopolamine gr. 1/150 but no general anaesthesia. 11 a.m. injection of " sodium iodide " solution. Patient returned to ward shortly before noon. One officer familiar with the previous cases and visiting the ward at the time observed the characteristic odour of garlic. The patient was fully conscious, very slightly cyanosed and felt well apart from pain in the left lumbar region. A second medical officer did not recognize the odour and ordered morphia gr. $\frac{1}{4}$ for the pain. 1.45 a.m. patient was confused but conscious, and markedly cyanosed. Pulse 90 regular. Respiration was normal. No other abnormal physical findings were detected. Blood count, red blood cells 4,900,000, haemoglobin 99 per cent., white blood cells 16,000, polymorphs 80 per cent., large lymphocytes 12 per cent., small lymphocytes 6 per cent., monocytes 2 per cent. Film was normal in all respects. Spectroscopy revealed bands of methaemoglobin. Continuous oxygen and intravenous fluids, 5 per cent. glucose with 3 per cent. sodium citrate, were ordered. The cyanosis was severe and affected the extremities, head, neck and shoulders. It was partially relieved by oxygen therapy, but returned and by 3 p.m. was of a blackish hue. The respirations were now irregular—rate 26-36. Pulse 134. BP 100 systolic and diastolic reading not obtainable. The cough reflex was strong. The patient was unconscious, but responded to painful stimuli. 4 p.m. There was slight improvement in general condition, but consciousness was not regained. Condition gradually deteriorated, with Blot's breathing, frothing at mouth, and deepening cyanosis despite intensive oxygen therapy. Death occurred at 5 p.m. Autopsy was performed within two hours of death.

Spectroscopic analysis of the Blood. A sample of blood from Case 3 was subjected to both spectroscopic and chemical analysis. It was observed at

once that the sample had a most peculiar and characteristic odour not unlike garlic.

Some haemolysis had taken place when the specimen was received, but the plasma was separated and examined with the Hartridge Reversion Spectroscope, using a sample 1 inch in depth. Bands were observed at 623 mμ and 540mμ. On addition of ammonium sulphide the band at 623 mμ was dispersed; with Stoke's reagent or molar. NaOH, though reduced in intensity, it was still faintly visible. A specimen of methaemoglobin prepared from fresh human blood when compared in the Reversion Spectroscope gave an absorption band at 628 mμ.

Chemical Analysis of the Blood. Chemical analysis demonstrated the presence of volatile alkyl telluride either ethyl or methyl telluride.

Chemical Analysis of the Liver, Spleen and Kidneys. Tellurium was present in all the above organs.

Necropsies were performed on Cases 1 and 3. They had in common the following striking features, marked cyanosis of the head and neck, the subcutaneous fat and the fat of the depots exhibited an intense yellow and the muscles a deep red brown colourization. The mucosa of the bladder and of the ureter on the side of the injection was blackened. The ureter which had not been catheterized was normal. The pungent characteristic odour noted before death and in the blood specimens was very obvious when both bodies were opened and increased in intensity as various organs were sectioned during the course of the examination.

There was in addition congestion of the lungs, liver, spleen and kidneys.

Histological examination of the material from the liver, lungs, spleen, heart, brain and kidneys of both cases showed no changes that might be attributable specifically to tellurium poisoning. The principal findings were fatty degeneration, oedema and congestion most marked in the liver. There were signs of ureteric irritation as evidenced by small aggregations of polymorphs in the subepithelial tissue.

Experimental

A 250 gm. guinea-pig was injected intraperitoneally with 2·5 ml. of a 0·1 per cent. solution of sodium tellurite representing a dose weight ratio of 1 in 10,000. There was no evidence of irritation during the injection and no ill effect over the next 24 hours. The next day 2·5 ml. of a 1 per cent. solution of sodium tellurite were injected intraperitoneally representing a dose weight ratio of 1 in 1000. There was some momentary local irritation at the time of the injection but the guinea-pig appeared quite comfortable when returned to its cage. Ten minutes after the injection it started gasping for breath, rolled over on its side, and died 13 minutes after the injection with slow gasping respirations. It was thought that, terminally, a faint suggestion of the characteristic odour could be detected about the cage. Autopsy was performed at once; as soon as the thoracic and peritoneal cavities were opened the characteristic odour was easily detected by everyone. There was a little milky lymph in the right paracolic gutter but the peritoneal cavity had lost none of its characteristic healthy sheen. No gross macroscopic nor microscopic lesions were detected in the organs. The blood was examined by the Reversion Spectroscope. There was a faint but definite band at 627/628 mμ which was dispersed by ammonium sulphide solution and Stoke's reagent.

Discussion

The source of the tellurium was traced to a bottle of sodium tellurite labelled "sodium iodide" and introduced into the body during the retrograde pyelography examination. In cases 1 and 3 the amount of tellurite introduced was considerable, 10–15 c.c. of a 12½ per cent. solution, i.e. approximately 2 gm. The clinical course following the introduction of the poison was similar in both cases, death occuring after approximately 6 hours. The symptoms in order of their appearance were the characteristic garlic odour, described by some observers as a mixture of garlic and carbide, renal pain on the side of passing of the catheter, cyanosis, vomiting, stupor, loss of consciousness, irregular breathing and death. In Case 2 owing to a defect in the ureteric catheter the amount of fluid injected was very small and no accurate estimate can be given.

Gies and Mead (1900) found that the experimental administration of sodium tellurite to dogs in doses not exceeding 0·1 gm. produced no ill effects apart from the development of the characteristic garlic-like odour. Larger doses 0·2–0·5 gm. per dog caused violent vomiting and disintegration of the gastric mucous membrane. In their experiments the drug was administered orally which may have accounted for the prevalence of vomiting. In the present series of cases vomiting was not a prominent feature. In fact in Case 2 owing to the prevalence of gastro-intestinal diseases in the ward it was impossible to exclude the latter as the cause of the vomiting. The epithelial lining of the ureter in the two fatal cases described was destroyed but in view of the absence of any necrotic changes surrounding the site of intraperitoneal injection of the drug into guinea-pigs it is probable that the destruction resulted from trauma during the passage of the ureteric catheter.

The clinical features common to the three cases were the garlic-like odour and cyanosis. Once experienced the odour is unmistakable and readily recognized, and was first noted by Hauser (1853) following the administration of potassium tellurite to dogs and to man but even trained medical personnel unfamiliar with the odour may easily overlook its presence, as occurred in Case 3. The isolation of a volatile telluride from the blood stream is of interest and may explain the characteristic pungent odour of the breath and of all the organs.

Assuming the weight of the subject to be normal—i.e. 70 kilos—the maximum dose absorbed would not give a higher concentration of telluride than 1 in 35,000. Man would therefore appear to be more susceptible to the toxic effect of telluride than the guinea-pig and the dog.

The initial examination of the blood by means of a simple spectroscope suggested that the cyanosis was due to the presence of methaemoglobin but examination with the Reversion Spectroscope suggested that the abnormal pigment was not methaemoglobin. The exact chemical composition of the pigment responsible for the cyanosis was not determined, although the intriguing possibility of some combination of tellurium with haemoglobin analogous to sulphaemoglobin cannot be excluded.

Summary

The clinical and pathological findings in three cases—two fatal—of accidental poisoning due to sodium tellurite are described.

Acknowledgment

We wish to thank Colonel N. Cameron, O.B.E., Officer Commanding the Hospital, for permission to publish these cases.

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SOME TOXIC EFFECTS OF VANADIUM PENTOXIDE

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Chemistry

Vanadium is a silvery-white metal of specific gravity 5·96, melting-point 1710° C., atomic weight 50·95, atomic volume 9·3, and atomic number 23. The name is derived from that of the Scandinavian goddess Vanadis (Parkes and Mellor, 1939). It is rarely encountered in its pure state owing to difficulties in production consequent upon its tendency to oxidation. In Mendeléef's Periodic Table the atomic number of 23 places it in the A Sub-Group of Group V, with niobium, tantalum and proactinium. The last-named substance has radio-active properties. From the electronic configurations of the atoms in members of this sub-group it is evident that they are transition elements, since the penultimate electron group is incomplete and is in process of filling up. As transition elements, they are characterized by very variable valencies, the formation of highly coloured ions, are paramagnetic and have marked catalytic power.

Four oxides are known: hypovanadous oxide (VO or V_2O_2), vanadous oxide or vanadium trioxide (V_2O_3), hypovanadic oxide (VO_2 or V_2O_4) and vanadic oxide or vanadium pentoxide (V_2O_5). Hypovanadous oxide is a black powder. With acids it produces lavender-coloured solutions of hypovanadous salts from which caustic alkalies precipitate hypovanadous hydroxide, $V(OH)_2$, one of the most powerful reducing agents known. Vanadous oxide is also a black powder and its salts are green in solution. Hypovanadic oxide forms blue solutions of vanadyl salts. Vanadium pentoxide is a yellowish red powder which is sparingly soluble in water. The basic properties of vanadium oxides diminish as the oxygen part of the molecule increases and the higher oxides exhibit acidic as well as basic properties.

Manufacture

The ores are distributed mainly in Peru, South Africa and Rhodesia, and occur as carnotite (potassium uranyl vanadate), patronite (impure vanadium sulphide) and vanadinite. In countries where the ores are not found, petroleum residues form the main sources. Three groups of raw materials are used in the plant about to be considered: (1) soot from oil burning liners, (2) furnace residues from oil refineries and (3) slags from the production of ferro-vanadium. The soot is ground and extracted with caustic soda solution and filtered. The filtrate contains the bulk of the vanadium as sodium vanadate. The filter cake is mostly carbon but contains some sodium vanadate which is recovered. The furnace residues are fused with soda ash. The roast is crushed, the dust containing about 15 per cent. V_2O_5, and extracted with water and filtered. Sodium vanadate is present in the liquor. Slags are crushed, dried, ground the dust containing about 10 per cent. V_2O_5 and roasted with soda ash. The roast is then crushed, ground, extracted with water and filtered. The liquor contains sodium vanadate. The sodium vanadate liquors are alkaline. They are neutralized with sulphuric acid and filtered to remove small proportions of alumina and silica. The last substance is apt to be present from the firebrick in the raw material. Sulphuric acid is again added to the neutralized liquors to precipitate vanadium, mainly as the hydrated V_2O_5. Filtration, drying, roasting and crushing complete the process. The oxide is reduced to ferro-vanadium by the alumino-thermite process. Dust arises mainly from the crushing of oxide and in the plant under consideration is now entirely a closed operation.

Uses

The chief uses of vanadium are to raise the hardness and malleability of steel and to increase its fatigue-resisting properties. For example, it is said that ½ per cent. of vanadium will raise the tensile strength of a sample of steel from 7½ to 13 tons per square inch. This property therefore commends its use to manufacturers of motor cars, Diesel engines and tools. The non-ferrous alloys are very hard and are resistant to atmospheric corrosion. The pentoxide is used as an oxidizing catalyst in the conversion of naphthalene to phthalic anhydride and in place of platinum in the modified contact process of manufacturing sulphuric acid. Indeed, in the latter process it is likely to supersede entirely the use of platinum. Compounds of vanadium are also used in the preparation of ink, in dyeing, as accelerators in the drying of paints and varnishes, in insecticides, photographic chemicals, in glass manufacture and therapeutic substances.

Pharmacology

Experimental poisoning in animals indicates similar symptoms to those observed industrially by Dutton (1911). These will be described in a later

section. Daniel and Lillie (1938) found that cumulation does not occur and that acute and chronic symptoms are similar. They administered by mouth sodium meta- and ortho-vanadate to rats and noted immediate intense distress, haemorrhages from the nose and intestines, marked diarrhoea, paralysis of the hind legs, dyspnoea and convulsions. The post-mortem findings were those of acute desquammative enteritis.

D. E. Jackson's (1912) experiments concerned the circulatory effects of the intravenous injection of sodium orthovanadate. There was a rise in blood pressure due to systemic vaso-constriction by direct action on arterial muscle or vasomotor endings. The heart was not strengthened. The pressure in the pulmonary artery also rose by systemic and pulmonary vaso-constriction. Perfused lung vessels were strongly constricted. Bronchial muscle also showed moderate but prolonged constriction.

Luzzato (1903) showed that the effects of vanadium chloride and bromide are the usual lesions of a metal in the kidneys, liver, stomach and intestines. He found no lesions in the central nervous system. The blood pressure was raised and the excitability of the vagus depressed. Death occurred from respiratory failure.

Fischer (1934) recorded that ' in experimental poisoning of a more or less serious character, the mucous membrane of the eyes, nose and stomach becomes covered with a greenish coating containing vanadium.'

Levaditi (1928) investigated the spirochaetal value of vanadium on rabbits and Pereira (1939) carried out a clinical research with the same object. The latter injected vanadium sodium tartrate, 0·15 gm. in 2 c.c. of water, intramuscularly, twice a week. He reported prompt disappearance of spirochaetes, secondary lesions and gummata which had been refractory to bismuth and arsphenamine. He found it also effective in framboesia. The toxicity was low: stomatitis, erythema and hepatic disorders were rare sequels.

Sollman's (1942) views on the therapeutic uses of vanadium are less optimistic, for he states that ' the pharmacologic actions do not point to any definite therapeutic usefulness. Vanadium has been tried as an alternative similar to arsenic, in tuberculosis, diabetes, etc., but the results are not convincing. It was administered as sodium metavanadate, a white, almost tasteless powder, soluble in water. The dose used was 1 to 8 mgm. ($\frac{1}{60}$ to $\frac{1}{8}$ grain).'

Industrial Poisoning

World consumption of vanadium more than trebled between 1936 and 1939. Poisoning from this source is, therefore, likely to be a factor of increasing importance (Fairhall, 1945). Dutton (1911) was the first to describe vanadium poisoning as he observed it in a plant in Carnegie, Pennsylvania where South American ore was used. He described a dry, irritating, paroxysmal cough, anaemia preceded by an increased red cell count, pallor, anorexia, emaciation, severe haemoptyses, albuminuria and casts, haematuria, fine tremours, failure of vision, vertigo, hysteria and melancholia as being the characteristic features. He found that tuberculosis was apt to supervene and that a fatal termination was not infrequent.

Hamilton (1943) has been unable to confirm Dutton's observations. She visited a plant where ferro-vanadium was manufactured from Peruvian ores by the alumino-thermite process and merely noted that the men suffered from a cough. At a mine in Colorado where the ore was found and then converted into sodium vanadate with salt, she was informed that the only hazard appeared to be chlorine which was liberated by the process. After precipitation with sulphuric acid, filtration and drying, the fine, sharp pointed crystals of sodium vanadate seemed to be very irritating to the nose, throat and bronchi. New men suffered most, from cough, but seemed to get acclimatized. Pneumonia was of the lobar type and was not noticeably prevalent. She had been informed of ' the complaint of twenty-two stevedores who were unloading Peruvian vanadium ore for only half a day and who insisted that the dust caused severe coughing which persisted through the night. There was, however, no time lost by these men.'

Symanski (1939) made a careful study of nineteen cases of vanadium poisoning which occurred among German metallurgical workers. He observed that only the workers themselves seemed cognizant of the health hazard. The effects appeared to be those of an acute irritant and were characterized by chronic bronchitis. With continued exposure specific infections might complicate the clinical picture. He recommended the mechanization and enclosure of all dusty processes.

The writer's experience extends to a period of nine years' observation of some 50 to 90 workers. At the present time about 50 persons are engaged, a dozen of whom are women. The latter are exposed to the same hazards as the men, but are more careful over personal hygiene and seem more resistant to vanadium poisoning. It should be added that women only came into this employment during the war years when mechanization began to displace the older, open, dusty methods. About 20 men have been engaged on the plant for periods in excess of eight years. As a class, they are paler and less robust in appearance than other process workers. The investigation of symptomatology was complicated by two factors: first, the presence of products of combustion of producer gas escaping into the department from the furnaces, a factor now in course of elimination, and secondly, the curious pride of workmen which impels them to exaggerate the risks of their occupation to new employees. It had been well known for some years that vanadium pentoxide dust caused bronchitis among the workers and efforts had been made to control the dust, but the full significance of the condition was not realized as the bronchitis always disappeared after an absence from the irritant of

10 to 14 days and occasional skiagrams did not suggest anything worse than might be expected from, say, the inhalation of carbon dust. It was the observation of Dr. M. H. Jupe in the case of one vanadium worker (case 7 Plate 3) of the presence of reticulation which stimulated further investigation. Other cases of reticulation were found and also certain systemic effects tending to confirm Dutton's observations in 1911, observations which have been consistently controverted ever since. It will be noted that no expression of opinion is made here on the questions of anaemia and renal impairment, as systematic blood counts and examinations of centrifuged urinary deposits have not been performed. All that can be said at this stage is that the skin is usually pale, but there are no clinical signs of anaemia and that one man exhibited hyaline and granular casts in his urine. Also no opportunity for post-mortem examination has so far occurred and the underlying pathology of the radiological features is unknown.

(*a*) *Dust reticulation and pneumoconiosis.* These terms have assumed restrictive connotations since the work of Hart and Aslett (1942) on South Wales coalminers and these narrow interpretations have been confirmed in the Pneumoconiosis Regulations of 1943 with the exception that the aetiology has been extended to other dusts. Hart and Aslett offered the following definition: 'A dust-ridden state of the lungs corresponding to x-ray reticulation, characterized by accumulation of silica and concommitant elaboration of reticular tissue throughout the framework of the lungs.' They considered the synonym 'anthracosis' to be unsuitable. The condition is due to the inhalation of coal-dust which contains a proportion of silica in its free and combined states; but whereas free silica causes nodulation and a minimum of reticulation, the predominance of silicates of the mica and clay types seems to cause reticulation and a minimum of nodulation. The carbon would appear to be a fortuitous factor. The pathology consists in a proliferation of reticulum fibres consequent upon the immobilization of dust cells in the intra-pulmonary lymphatics. It is not necessarily progressive. This definition, useful though it may be for certain purposes, is neither in lineal descent from Aschoff's conception of a vitally staining reticulo-endothelial system, nor is it strictly derivative from Maximow's histological classification of tissues forming an argyrophil, undifferentiated mesenchyme or syncytium. But what is more to present purposes is that it excludes similar radiological appearances due to substances other than dust containing coal and siliceous materials unless pulmonary fibrosis is a known concommitant.

But reticulation is a radiological appearance which is by no means restricted to the inhalation of carbonaceous and silicate dusts; indeed, it is often found among city dwellers as a class as well as in occupations exposing workers to certain other specific dusts. The first radiological phenomenon to be observed in all workers exposed to dusty processes is a continuation of the linear markings towards the peripheries of the lung fields. Whether this is due to fibrotic changes in the endopulmonary lymphatics or merely to shadows cast by the dust itself in these channels is generally a matter of opinion; undoubtedly the amount of fibrosis, if any, depends upon the character of the dust. As the perivascular lymphatics of the smaller vessels become involved in the process the lines seem to become interlaced and give the appearance of a network. Emphysema then occurs as a compensatory factor. Although no precise clinical phenomena distinguish the two stages, it may be stated generally that catarrhal features characterize the phase of increased linear striations and dyspnoea and a harsh and prolonged expiratory murmur accompany the more advanced stage. Vanadium workers complain of paroxysmal cough, chiefly troublesome on wakening, tenacious sputum very occasionally blood-stained and pains in the chest.

(*b*) *Systemic intoxication.* Although there are probably other dusts besides vanadium which cause reticulation of the lungs as well as systemic poisoning, at present the position of vanadium in these respects seems to be unique. Bronchospasm is commonly present and the blood pressure is frequently raised. These findings are in accord with the experimental work of Jackson. Palpitation on exertion, such as climbing a flight of stairs, is also consistent with Luzzato's (1903) observation that the irritability of the vagus is depressed. An accentuated second pulmonary sound which is occasionally found, seems to confirm the experimental evidence that the pressure in the pulmonary artery is elevated. Pallor of the skin, which is fairly characteristic, may be ascribed to the direct constricting action of vanadium on arterial muscle and myoneural junctions. No case of clubbing of the fingers or toes has been noted. Many exhibit a greenish-black discoloration of the tongue, more particularly men with septic teeth and furred tongues. Withdrawal from contact with the substance suffices to cause the disappearance of the discolouration in two to three days. It cannot be removed by cleaning and may be due to reduction of the pentoxide to the trioxide and formation of green salts by the actions of ptyalin and acid-forming bacteria in the mouth. It is probably evidence of exposure, rather than of intoxication (Case 9). The workman complains of a salty taste. Tremor of the fingers is fairly common in those workers with prolonged exposures, is fine, but may affect the arms and be so coarse as to interfere, for example, with the act of recording the blood pressure. Diarrhoea, anorexia, emaciation, visual defects, paralysis, vertigo, convulsions, hysteria and melancholia have not been observed. No case of tuberculosis has occurred, but three cases of lower lobe pneumonia (one fatal) and one case of middle lobe pneumonia are known. Workmen complain of frequent attacks of coryza.

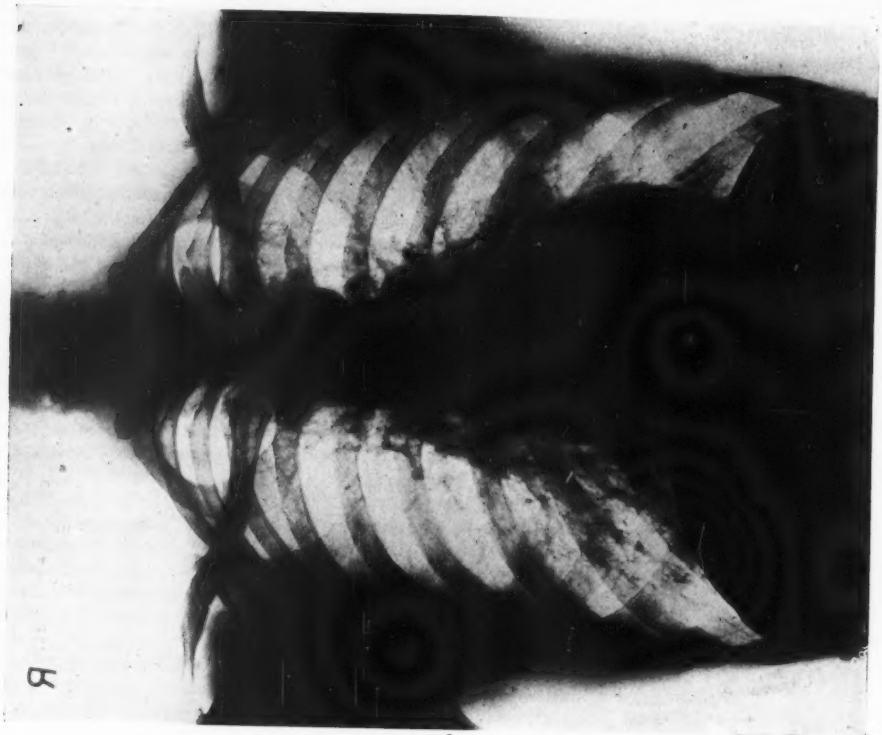

FIG. 2.—X-ray of Case No. 6.

FIG. 1.—X-ray of Case No. 1.

Illustrative Cases

Case 1. A. H. W., male, aged 30. Vanadium worker 1938 to 1946. He complained of cough, dyspnoea on exertion or whilst wearing a gas-mask. His weight on 30/3/38 was 9 st. 11 lb., on 28/4/45 9 st. 10½ lb. On examination, he had many rhonchi throughout his chest. X-ray showed increased linear striations (fig. 1).

Case 2. A. H., male, aged 45. Chargehand Vanadium Dept. 1930 to 1946. He suffered from attacks of lobar pneumonia in 1937 and 1940. He complained of cough and dyspnoea. On examination, he was found to have a typical emphysematous chest. There were râles of all kinds in all areas. He showed tremor of his hands. X-ray revealed increased linear striations.

Case 3. E. J. M., male, aged 63. Vanadium worker 1941 to 1944. He complained of cough, of the stuffy, bronchitic type, with pains in both sides of his chest, and foul-smelling, bloodstained, copious sputum. On examination (24/6/42 and 13/3/43) he had a few high-pitched rhonchi at both bases. X-ray revealed no abnormality. He died of lobar pneumonia in 1944.

Case 4. C. A. R., male, aged 35. Vanadium worker 1938 to 1945 and then transferred. No antecedent history of note. He developed a pneumonia of the right mid lobe on May 17, 1945, during which he appeared very toxic and was tender over his liver. His blood-pressure was 130/84. An effusion appeared at the right base which was absorbed satisfactorily. On recovery, the skiagram indicated reticulation in both mid lung fields.

Case 5. B. T. W. J., male, aged 53. Vanadium worker 1944 to 1946, engaged in emptying ore from bags into cannisters. He complained that Rhodesian ore (mainly V_2O_4) made his cough worse. In 1943 he had lobar pneumonia, and he had further attacks of pleurisy in March and July, 1945. On examination, he had many râles throughout both lungs and an accentuated pulmonary second sound. His blood pressure was 170/100.

Case 6. J. P., male, aged 41. Vanadium worker 1935 to 1945. He complained that he began to suffer from a paroxysmal cough about a month after his employment on vanadium and it continued all the time he was in the department, except when on holiday. Latterly, it was accompanied by severe and troublesome dyspnoea and sharp chest pains, particularly on coughing and deep breathing. His weight on 31/8/38 was 9 st. 10¼ lb.; on 22/5/43 9 st. 2¾ lb. His blood pressure was 150/100. He had an accentuated pulmonary second sound and scattered râles throughout both lungs. X-ray showed reticulation in left mid zone and right base. There was slight enlargement of the left ventricle (fig. 2).

Case 7. W. J. C., male, aged 38. Vanadium worker 1940 to 1946. No antecedent history of note. He complained of cough all the time he had been in the department. He sometimes felt a sharp pain in the region of left nipple on coughing or breathing deeply. Dyspnoea and a feeling of exhaustion commenced about March, 1943. Cough was mainly troublesome on rising, but sometimes on going to bed. His sputum did not come up easily, was whitish, tasteless and inodorous. He was seldom free from nasal catarrh. He had attacks of pleurisy in 1941. His weight on 2/11/39 was 10 st. 5¼ lb.; on 25/4/46 10 st. ¼ lb. On examination, he had a pale skin; the mucous membranes were, however, well coloured. His blood pressure was 148/94. He had an accentuated pulmonary second sound. There were many rhonchi throughout the chest. X-ray showed reticulation (fig. 3).

Case 8. C. J. L., male, aged 61. Vanadium worker August, 1940 to May, 1941. He had a history of

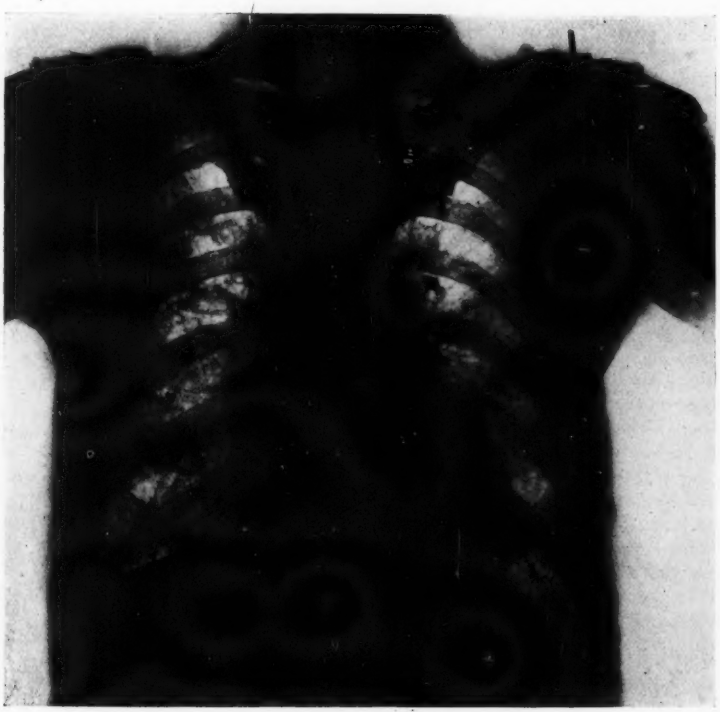

FIG. 3.—X-ray of chest of Case No. 7, showing reticulation.

bronchitis since 1920. Dyspnoea became very severe during his exposure to vanadium dusts, improved considerably after transfer, but has recently returned. It was brought on by exertion such as walking. On examination, he had a typical emphysematous chest. There were râles of all kinds in all areas, and he had a harsh and prolonged expiratory murmur. His blood pressure was 140/94. X-ray showed reticulation in both upper lung fields and emphysema at the bases.

Case 9. M. J. R., male, aged 31. Vanadium worker for 5 months. He complained of cough for 4 months and a salty taste in mouth. He had a greenish-black tongue. His blood-pressure was 132/90. There was no abnormal physical signs in his heart and lungs.

Case 10. C. R., male, aged 47. Vanadium worker 14 years. No antecedent history of note. He complained of paroxysmal cough chiefly on rising, but also on retiring. He coughed up tenacious, grey, tasteless, inodorous sputum, and had a salty taste in his mouth. Dyspnoea at end of shift was sometimes extreme and he had to lie down. He always had difficulty in getting home for the same reason. His chest felt tight, and there was palpitation on exertion, such as going up a flight of stairs. On examination, he had a greenish-black tongue. His blood pressure was 120/80. He had a coarse tremor of both hands. There were many rhonchi throughout both lungs.

Summary

An industrial disease is described with the unusual combination of dust reticulation and systemic intoxication. The observed features of the syndrome associated with industrial exposure to vanadium pentoxide are: (1) pallor of the skin, (2) greenish-black discoloration of the tongue, (3) paroxysmal cough, rarely with haemoptysis, (4) dyspnoea, (5) pains in the chest, (6) palpitation on exertion, (7) tremor of the fingers and arms, (8) barrel-shaped, emphysematous chest, (9) profuse bronchitis, (10) bronchospasm, (11) raised blood pressure, accentuated pulmonary second sound, and (12) reticulation on X-ray examination. It seems likely that colds and pneumonia are of more frequent occurrence than in the general population.

Ten case histories are briefly reproduced.

Acknowledgements

I am grateful to Messrs. Murex, Ltd., for giving the facilities to observe this condition and for their encouragement throughout the work. I have to thank Dr. M. H. Jupe for permission to publish the X-ray of Case 7.

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TOXIC MANIFESTATIONS OF OSMIUM TETROXIDE

BY

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Osmium is one of the precious metals and was discovered in 1803 by Tenant. It occurs naturally in close association with iridium. Osmiridium is an exceptionally hard alloy with a high melting-point (2700° C.) and for this reason it is used extensively for tipping fountain-pen nibs. It is also used for electrical contacts, as a catalyst in the preparation of synthetic ammonia, and for measuring the rapidity of explosion of gun-cotton. Before the introduction of tungsten, electric lamp filaments were sometimes made with it. Osmium is also used in the taking of finger-prints, and osmic acid which is a watery solution of osmium tetroxide is used for activating solutions of chlorate of potassium; hardening and colouring certain preparations and in histology for the fixing and staining of nerves and fat. The precious metals are likely to be used increasingly in the future and it is felt that the possible health risks in the use of their derivatives should be known at the outset.

Review of Literature

Metallic osmium is innocuous but osmium tetroxide is slowly formed on exposure of the spongy metal to air. The toxic properties of osmium tetroxide were first noticed by Gmélin (1827) who described the occurrence of vomiting when it was given by mouth. Intravenous injections in animals caused serous exudate from the lungs, three such injections producing paralysis and death. Vauquelin (1814), Berzélius (1828) and Wohlea (1834) drew attention to its corrosive action on the respiratory tract and eyes. Branell (1849) suggested that the symptoms resulted from the oxidizing action of osmium tetroxide (OsO_4) which is reduced to osmium dioxide (OsO_2), which is then hydrated ($OsO_2 2H_2O$). Deville and Debray (1859, 1874) described how one of them was rendered almost blind for 24 hours when a current of air taking away vapour was thrown back unexpectedly on to him. It produced a sensation like a vigorous blow on the eye. Osmium tetroxide on the conjunctivae produced inflammation which continued to render sight troublesome for some weeks. The author also developed mild asthmatic symptoms and a co-worker a painful skin eruption. Raymond (1874) reports the only fatal case in the literature. The patient was employed by Vulpain in Paris and the vapours of osmic acid gave rise to a capillary bronchitis from which he died. Necropsy revealed a confluent broncho-pneumonia with a tendency to suppuration and gangrene; there was also a fatty degeneration of the epithelium of the renal tubules. Bardieux (1898) showed that the subcutaneous and intramuscular injection of a 1 per cent. solution of osmic acid produced no serious effect in animals but that small quantities injected into the lungs were immediately fatal. Attempts have been made to make use of the solution in treating diseases such as tuberculosis (Walbum, 1926), syphilis (Levaditi, 1927; and Jahnel, 1937) and cancer (Kraus, 1931) but without success, though Fischl (1933) showed it possessed some spirochaetocidal properties in experimentally infected mice Coca (1908) studied the effect of the substance on red blood cells, but the most comprehensive study of the toxicological properties of osmium tetroxide is by Brunot (1933). He exposed himself to some of the vapours and after 10 minutes he noticed a metallic taste in the mouth and found that smoking was unpleasant; after 30 minutes he noticed a smarting sensation in his eyes, lacrimation which was exaggerated by efforts to read; 3 hours later reading was difficult, he felt a definite constriction in the chest and found breathing also difficult; on going out into the street the lamps appeared to be surrounded by large haloes as if there were a dense fog. He therefore carried out animal experiments. Rabbits were exposed for 30 minutes in a box of 190 litres capacity. An ampoule containing 250 mg. of osmium tetroxide was placed in it on a hot plate and a fan was used to distribute it throughout the atmosphere. During the exposure the tetroxide was steadily reduced to the dioxide and metal so that at the end of the experiment only 10 per cent. was left. The muzzles of the animals became blackened, the pelt changed from white to dirty grey, and a brown film developed over the sclera and cornea. After 4 days the animals showed marked pulmonary embarrassment and when killed the lungs were voluminous and did not float in water. They were dark red and solid with scattered irregular purple areas and the bronchi were filled with pus. The kidneys showed cloudy swelling and granulation of the epithelium. After exposure to 500 mg. and 1 g. the animals became semi-comatose a few minutes after the

exposure, showed acute bronchial irritation and pulmonary embarrassment and died within 30 hours. He also placed 1 drop of 1 per cent. osmium tetroxide in water into the conjunctival sacs of rabbits. After 24 hours there was oedema and swelling of the lids with profuse purulent discharge and a metallic brown stain on all parts of the conjunctiva. After 48 hours the cornea was milky and semi-opaque with superficial conjunctival ulcers. Ten days after the exposure the acute stage had subsided, but there was corneal opacity and a pannus began to form which after 1 month slowly extended towards the centre of the cornea.

Recent Investigation

We had the opportunity of observing the effect of osmium tetroxide on seven men who were engaged in refining osmiridium. The findings appeared worth reporting. The atmospheric concentration of osmium tetroxide to which the men were exposed was estimated. This part of the investigation is described first, the clinical findings being given later.

Environmental Study

In dissolving ores containing the precious metals with aqua regia, osmium compounds are apt to gain access to the surrounding atmosphere in the fine spray which is given by the acid reaction. Samples of the air near to the action pots were obtained by sucking the air through a cone-shaped filter which was fitted with a low-resistance Whatman filter paper. Suction was obtained by means of an injector pump attached to an air line, and in the system was incorporated a flowmeter reading up to 100 litres per hour. Three 2-hour samples were taken during the reaction and distillation processes and estimated by the following method.

The paper containing the osmium in particulate form was gently boiled under reflux with 2·5 ml. of concentrated nitric acid for half an hour. Water was then added to the macerate until the solution was 10 per cent. with regard to nitric acid, and the volume was then about 25 ml. This solution was then transferred to an all-glass microdistillation apparatus and gently distilled into a receiver containing 3 ml. of concentrated hydrochloric acid saturated with sulphur dioxide. When about a fifth of the solution (5·5 ml.) has been distilled, the distillation was terminated as all the osmium was by then removed. The solution in the receiver was estimated colorimetrically in the following way. To the solution was added 0·1 ml. of 10 per cent. stannous chloride solution in 10 per cent. HCl, 1 ml. of 2 per cent. gum ghatti solution, and 0·5 ml. of 10 per cent. aqueous thiourea solution. The mixture was then treated in a boiling water bath for 5 minutes, cooled and the pink colour which developed was read in the Spekker photoelectric absorptiometer fitted with a green filter. The result was referred to a calibration graph which was prepared by submitting standard solutions of osmic acid to a similar colorimetric technique. Alternatively a measurement can be made using a colorimeter if standard solutions of osmium are used in the comparison cell. Where very small quantities of osmium are encountered, a micro-technique may be employed, in which case the distillate may be arranged so that the osmium is contained in 2 ml. of liquid and colour development carried out as above, making certain that the concentration of acid in the distilling solution is of the order of 10 per cent. by volume. The colour is read in the Spekker absorptiometer fitted with micro cups, or in a microcolorimeter. This refinement was carried out when the osmium present was appreciably lower than 100 μg.

CALIBRATION CURVE

μg. osmium	Spekker reading
0	0
100	0·070
200	0·145
300	0·225
400	0·300
500	0·375
600	0·450
1000	0·750

ATMOSPHERE SAMPLES

(2-hour sample at 75 litres per hour = 150 litres)

1. Osmium found = 20μg. 133μg per cu. metre.
2. Osmium found = 70μg. 467μg per cu metre.
3. Osmium found = 96μg. 640μg per cu. metre.

Clinical Picture

The men exposed to these concentrations had symptoms similar to those described by Brunot (1933). They had irritation of the eyes with pain and lacrimation. They saw large haloes round all lights, one man saying they were green in the centre and red round the outside. Mostly the symptoms subsided within 24 hours. One of the men complained of bronchial irritation which caused a cough and thick sputum. The case histories of the seven men are as follows:

Case Histories

Case 1. A. A., aged 33, had during the whole of his working life been an analytical chemist in contact with osmiridium, for 12 years with one firm and for the last 4 years at the firm where these observations were made. He said that when he was in contact with osmium tetroxide, he notice his eyes were sore and ran freely and that if he looked at a light there was a large halo round it. He had a frontal headache. The symptoms usually wore off during the night and he was normal the next morning. He frequently had small ulcers in his mouth. He had no other symptoms. His only previous illness was scarlet fever as a child. Apart from a slight deformity of his chest he had no abnormal physical signs; blood pressure, 130/80; urine acid, specific gravity 1012 and contained no albumen or sugar. Blood count showed 5,950,000 red cells; 118 per cent. haemoglobin (alkaline haematin-photoelectric cell method 13·8 g. of haemoglobin = 100 per cent.); 7000 white cells; 60 per cent. polymorphs, 36 per cent. lymphocytes, 1 per cent. eosinophils and 3 per cent. monocytes.

Case 2. T. T., aged 45. Before the 1914–18 war he worked in an office. During that war he served in the army and since then he had worked in the chemical

factory under observation, during the last 7 years in contact with osmiridium. He noticed a smarting and running of his eyes and slight frontal headache. Later there was a gritty feeling in the eyes and 'conjunctivitis.' A halo appeared around everything he used, and he was unable to see the picture at the cinema. The headlights of an approaching car would appear to take up the whole road. He had no other symptoms, no previous illnesses, and physical examination revealed no abnormal physical signs; blood pressure, 140/98; urine acid, specific gravity 1030, and contained no albumen or sugar. Blood count showed 5,600,000 red blood cells; 120 per cent. haemoglobin (alkaline haematin-photoelectric cell); 6200 white cells; 60 per cent. polymorphs, 34 per cent. lymphocytes, 2 per cent. eosinophils and 4 per cent. monocytes.

Case 3. F. R., aged 53. He had been a gardener, and served in the army before joining the firm under observation. He had worked in contact with osmiridium for 18 years. When he went home after working with osmiridium, he noticed a halo around the moon and the stars and all other lights. His eyes smarted but did not run. He had no headache, and the next morning his eyes felt quite normal. He had no other symptoms, and had had no illnesses apart from trench fever during the last war. He had no abnormal physical signs; blood pressure, 140/110; urine acid, specific gravity 1024, and contained no albumen or sugar. Blood count showed 5,000,000 red cells; 106 per cent. haemoglobin (alkaline haematin-photoelectric cell); 6000 white cells, 46 per cent. polymorphs, 49 per cent. lymphocytes, 4 per cent. eosinophils and 1 per cent. monocytes.

Case 4. J. K., aged 34. He had done general office work before joining this chemical firm 11 years ago. He had worked in contact with osmiridium during the past 18 months. He noticed that the osmium irritated his eyes and made them feel gritty. He had not noticed any running of the eyes. After the fusing of a charge, he noticed that there was a halo around all lights; these haloes had not disappeared by the next morning. He had no headache. He had no other symptoms. He had had measles and whooping cough as a child. On examination, apart from a small fatty hernia of the linea alba, there were no abnormal physical signs. His blood pressure was 110/80. The urine was acid, specific gravity 1020, and contained no albumen or sugar. His blood count showed 5,200,000 red cells; 110 per cent. haemoglobin (alkaline haematin-photoelectric cell); 13,000 white cells; 70 polymorphs, 26 per cent. lymphocytes, 1 per cent. eosinophils and 3 per cent. monocytes.

Case 5. E. J., aged 21, had worked at the firm for 5 years and had been in contact with osmiridium for 3 years. If the vapour of the tetroxide enters the workroom, it smells like garlic, it takes his breath away and produces irritating pains in the chest. It is difficult to breathe and he gets a cough with purulent sputum. He develops a halo around all lights, and this is green in the middle and red around the outside. The symptoms arise about two hours after contact and disappear after a night's sleep. He had no diplopia and no headache. He has no other symptoms; and apart from a mastoid operation 2 years ago, he has had no illness. He had no abnormal physical signs. The blood pressure was not taken and the urine was not examined. Blood count showed 5,800,000 red cells; 118 per cent. haemoglobin (alkaline haematin-photoelectric cell); 6400 white cells; 64 per cent. polymorphs, 34 per cent. lymphocytes, 1 per cent. basophils and 1 per cent. monocytes.

Case 6. F. T., aged 52. Apart from 4½ years' service n the army during the 1914–18 war, he had always worked at this firm. For 23 years he had been in contact with osmiridium. He had noticed a halo around all lights and a running of his eyes after he had been in contact with the substance. He could not read as the print appeared blurred. He had no headache. The symptoms had usually passed off by the next morning. He had no other symptoms and had not had any previous illnesses. He had no abnormal physical signs; blood pressure, 120/100; urine was acid, specific gravity 1020, and contained no albumen or sugar. Blood count showed 5,750,000 red cells; 116 per cent. haemoglobin (alkaline haematin-photoelectric cell); 8400 white cells; 68 per cent. polymorphs, 27 per cent. lymphocytes, 1 per cent. eosinophils and 4 per cent. monocytes.

Case 7. C. M., aged 48. He had worked all his life with the same firm, and for the past 8 years had been in contact with osmiridium. If he handles osmium tetroxide, it causes his eyes to smart for the rest of the day and even the day following, but they do not water. He sees a halo around all lights and has a severe headache behind the eyes. He had had no previous illness, and showed no abnormal physical signs; blood pressure, 120/100; urine was acid, specific gravity 1020, and contained no albumen or sugar. Blood count showed 4,980,000 red cells; 108 per cent. haemoglobin (alkaline haematin-photoelectric cell); 5800 white cells; 64 per cent. polymorphs, 32 per cent. lymphocytes, 1 per cent. eosinophils, 1 per cent. basophils and 2 per cent. monocytes.

Eye Symptoms

None of the seven men had eye symptoms at the time of the clinical examinations because they had not been working with osmiridium for some days. One case had a slight blepharitis; otherwise no abnormalities were detected. Arrangements were made for the men to be seen by Mr. A. B. Nutt, Ophthalmic Surgeon to the Sheffield Royal Infirmary, if at any time they had the characteristic eye symptoms. Two men went for examination and their eyes were examined with the slit-lamp microscope. On the cornea there appeared to be a number of minute doubly refractile areas, but the magnification of the microscope was not high enough for these areas to be identified with certainty. It was thought that they probably consisted of deposits of metallic osmium. Since then no cases of eye trouble have occurred because the fume of osmium tetroxide is now being removed from the workroom by mechanical ventilation.

Two men from the factory, however, volunteered to expose their eyes to the fume of osmium tetroxide and to undergo examination with a higher-powered slit lamp microscope. Nutt reported that the experiment was that of a man who brought some osmium and vaporized it in a small crucible held over a Bunsen flame; eye symptoms were produced in a very few minutes and again fine sparkling particles were noted on the surface of the cornea. If these were osmium crystals they might be expected to be very irritant. The appearance of these minute deposits of osmium on the cornea, even under high power, is however inconclusive; they sparkle as if they were crystalline but on examining them under a microscope nothing resembling osmium in a crystalline form could be found.

The characteristic symptoms are (i) a sensation of intense and sudden smarting of the eyes associated always with lacrimation and sometimes with frontal or orbital headache. (Histologists who use osmic acid as a myelin and fat stain are familiar with the headache which comes on if the bottle is

left unstoppered on the bench.) In some cases there is a gritty feeling in the eyes together with injection of the conjunctivae. All seven of our cases vividly described the halo which appears round lights. They were unable to read or visit the cinema during the height of the symptoms.

Nasal and Respiratory Symptoms

The fume of osmium tetroxide has a sudden vigorous effect on the mucous membranes of the nose, throat and bronchi and has the characteristics of an irrespirable gas. It has a ' kick like a mule.' If fairly high concentrations are inhaled there is a sense of momentary constriction of the chest and inability to breathe. We tested this for ourselves by inhaling the fume coming from a bottle containing osmic acid (or a solution of osmium tetroxide). The sensation of irritation of the nose, throat and chest persisted for some 12 hours after only one such inhalation.

Of our seven cases, however, only one complained of cough with purulent sputum, and he had been working with osmiridium for 3 years.

Prevention and Treatment

The symptoms can be prevented by not allowing the fume of osmium tetroxide to enter the air of the workroom. When a new ventilating plant was installed over the reaction vessels at the factory where these men were employed no further symptoms were observed.

In most cases the symptoms subsided within 24 hours and were not severe enough to require treatment. But where treatment is necessary it should be directed to preventing infection. The sulphapyridin group of drugs or penicillin should be used for chest symptoms and a propamidine eye lotion for any conjunctivitis that might develop. Claus (1848) suggested the inhalation of hydrogen sulphide (H_2S) as an antidote and his suggestion has been repeated in many text-books. Obviously, such a ' cure ' would be worse than the disease.

Summary

An account is given of a syndrome caused by the fume of osmium tetroxide evolved during the refining of osmiridium. It consisted of vigorous irritation of the conjunctivae and the mucous membranes of the nose, throat and bronchi, associated in some cases with frontal or orbital headache. The ' halo round lights ' was a characteristic symptom resulting from the irritation of the eyes. It was temporarily disabling in that the worker was unable to read for about 24 hours. The men were exposed to concentrations of osmium tetroxide in the atmosphere up to 640μg per cubic metre. No chronic or cumulative effects were noted. Full details are given of the method used for the quantitative estimation of osmium.

We are indebted to the Sheffield Smelting Co. for allowing us to carry out this investigation, and to Mr. L. J. Young for his help and co-operation during the time the work was carried out. We also wish to thank Mr. A. B. Nutt for his valuable and willing co-operation in the investigation of the eye symptoms.

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AN APPARATUS FOR THE MAINTENANCE OF A CARBON DUST CLOUD OF CONSTANT CONCENTRATION

BY

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During the course of an investigation of the health risk due to charcoal dust, it became necessary in connection with animal experiments to produce and maintain a cloud of charcoal containing a trace element of concentration and particle-size corresponding to that found under factory conditions. Animals were to be placed in the cloud for a considerable daily period extending over some months, and in order to estimate the dosage applied it was necessary to control the cloud concentration automatically. An apparatus was developed to fulfil these conditions, and as it may have a wider application for the purpose of maintaining a constant concentration of many other types of particulate cloud over a long period, it is described in detail. The physiological work has been completed and is the subject of an internal report of the Ministry of Supply.

Considerations of Cloud Production Method

The accurate determination of the optical size distribution of a cloud of carbon particles in which a considerable degree of aggregation exists is a matter of extreme difficulty and uncertainty. The cascade impactor was used as a comparative instrument, samples being taken of the factory cloud, the relative densities of the deposits on the four graded plates being used as standards. Samples of the experimental cloud could then be taken and compared with the standards, and the particle-size of the cloud adjusted in the appropriate manner. The apparatus used, therefore, had to fulfil the following conditions :

(*a*) to simulate the factory cloud the particle-size range must be such that when the cloud is sampled with the cascade impactor, at a flow-rate of 17·5 litres per minute, almost all the deposit is found on the third and fourth plates.

(*b*) sufficient cloud must be produced to maintain a concentration of 250 mgm. per cu. m. of carbon in a chamber of 7·8 cu. m.

(*c*) the apparatus should be capable of maintaining the cloud for at least 7 hours before recharging with charcoal is necessary.

(*d*) The apparatus should be simply controllable in order to facilitate the application of a suitable regulating device to ensure constancy of cloud concentration.

(*e*) In order to ensure the continuity of the experiment from the physiological viewpoint the apparatus must be comparatively trouble-free over a period of 700 hours at 7 hours per day on a 6-day week basis.

Method of Cloud Production

The apparatus finally developed is shown in fig. 1. The cloud is produced by the attrition of charcoal in the rotating drum, which is enclosed by the hood, and is then blown into the cloud chamber as shown by the arrows on the figure. The drum consists of a cylinder of 8 inches diameter and 10 inches length, the side being made of 22 mesh 26 S.W.G. copper gauze, and the end plates of $\frac{1}{8}$ inch thick sheet iron. Inside the drum the three radial vanes pour the charcoal and produce attrition as the drum rotates. The drum rests upon rubber-covered rollers, one of which is rotated by means of an electric motor. The second roller is merely an idle rotating support. The enclosing hood is fitted with an air intake and exit, the former being connected to the centrifugal blower drawing its supply of air from the cloud chamber. The exit rises vertically for 9 inches and is then led horizontally into the cloud chamber. This vertical rise is designed to remove the larger particles by elutriation, and the horizontal section performs the same function by sedimentation. Since the air supplied to the drum is derived from the cloud chamber and therefore contains considerable carbon cloud, the drum mechanism is enabled to maintain higher concentrations in the cloud chamber than would otherwise be possible, i.e., the system is regenerative. The drum is taken out through one side of the hood, which is detachable, and charged with charcoal after the removal of one end plate. The normal charging of granules is approximately one-third of the volume of the drum, the granules being of the order of 1·84 mm. in diameter. Debris from the ground charcoal is sieved through the side of the drum and accumulates on the floor of the hood ; this has to be removed at about 20-hour intervals.

Particle-size Distribution. As described in the previous paragraph, favourable conditions are obtained for the removal of the larger particles in the cloud by elutriation and sedimentation. A comparatively sharp cut-off is obtained on the maximum particle-size by control of the air stream

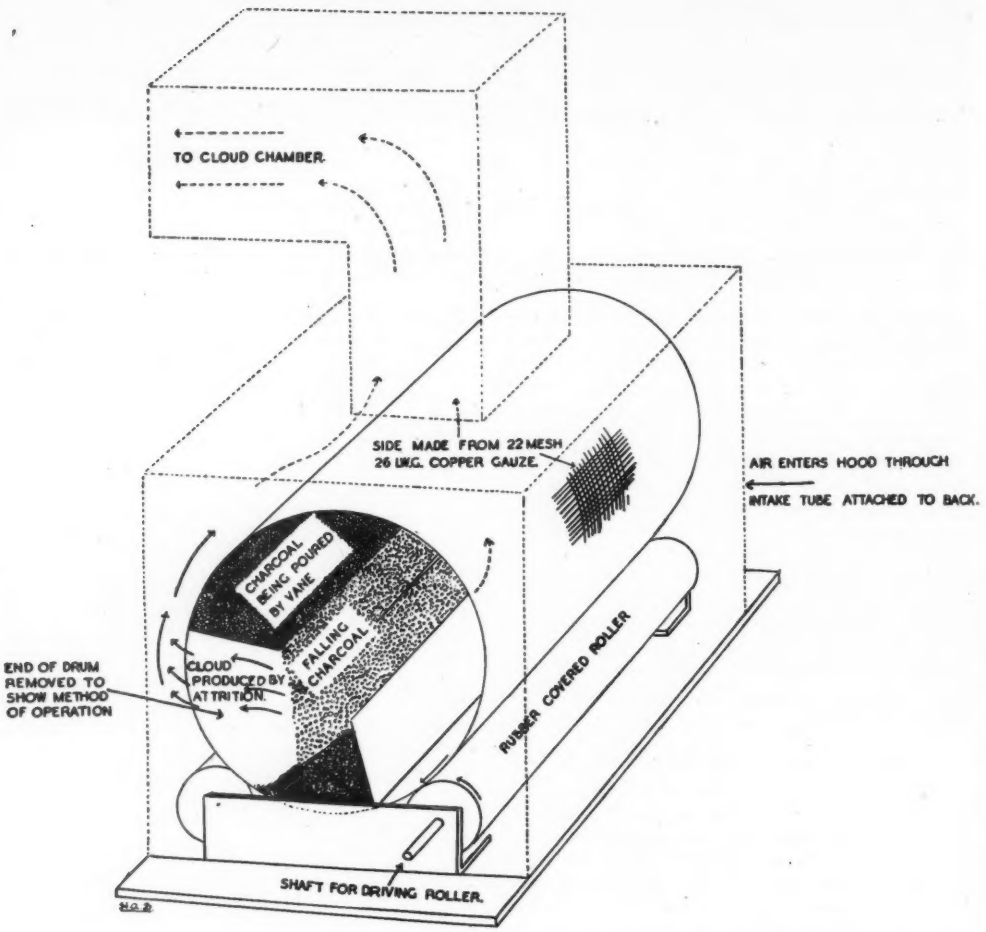

TO CLOUD CHAMBER.
SIDE MADE FROM 22 MESH 26 SWG. COPPER GAUZE.
AIR ENTERS HOOD THROUGH
INTAKE TUBE ATTACHED TO BACK.
CHARCOAL BEING POURED BY VANE
FALLING CHARCOAL
CLOUD PRODUCED BY ATTRITION.
END OF DRUM REMOVED TO SHOW METHOD OF OPERATION
RUBBER COVERED ROLLER
SHAFT FOR DRIVING ROLLER.

FIG. 1.—Diagram showing drum and roller. (Hood shown in dotted lines.)

velocity, varied by adjustment of a series resistance in the centrifugal blower circuit. This was adjusted until an accurate simulation of the size distribution of the factory cloud was obtained.

Control of Output. Since for the purpose of animal experiments the required cloud concentration was about three times the factory peak conditions, and since it was also desired to use only one drum for economy and simplicity, it was essential to achieve the maximum output. As the speed of rotation of the drum increases there is a progressive rise in output due to increasingly violent pouring and churning of the charcoal granules. On reaching some critical speed, centrifugal forces begin to take control and prevent the pouring from taking place, the system tending to rotate as a rigid whole, and causing a rapid diminution in output. Fortunately, the onset of the centrifugal effect is made apparent by a change in the sound emitted by the drum. It was therefore a simple matter to adjust a series resistance in the motor circuit to give to optimum speed of rotation for maximum output. A difficulty arose due to the drum motor being unable to start unaided from rest with the resistance adjusted as described above. Since regulation of cloud concentration was achieved by automatic switching of the drum motor at frequent intervals, this difficulty had to be overcome, and was obviated by temporarily short-circuiting the control resistance on starting. The device used is shown in fig. 2. When the regulator closes S_1, current flows directly to the motor through the closed mercury switch S_2. At the same time the solenoid is energized and withdraws the iron plunger from the mercury, and immediately mercury begins to flow from the reservoir, through the capillary, equalizing the level in the two arms. Conditions can be arranged so that after 10 to 12 seconds the level falls below the upper contact S_2, thus breaking the circuit.

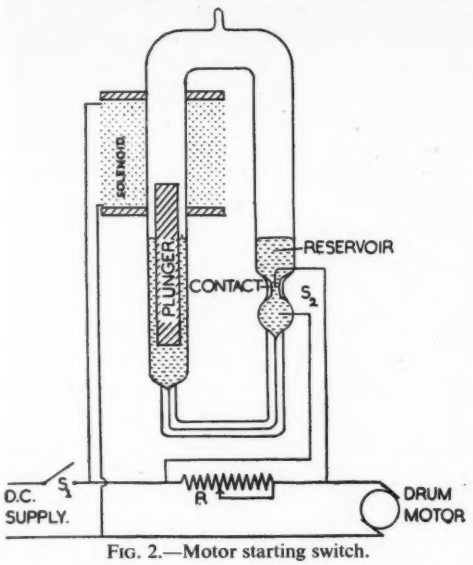


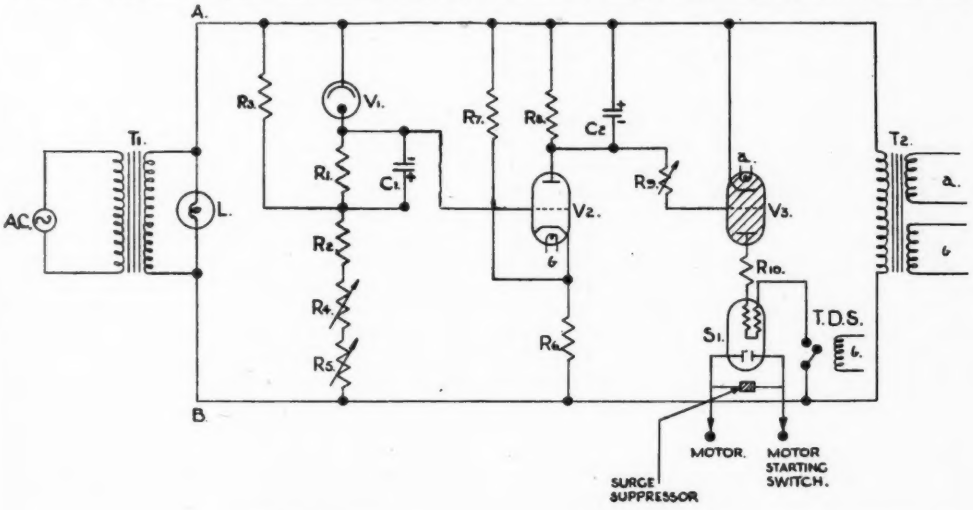
FIG. 2.—Motor starting switch.

The motor then receives its supply through the control resistance R, and this state continues so long as S_1 remains closed. On S_1 opening, the plunger falls, closing S_2, and the device is reset for a fresh cycle of operation.

The Requirements for the Regulator

For the purpose of physiological experiments, and having due regard for the probable accuracy of routine chemical estimation, it was decided that stabilization of cloud concentration to within ± 5 per cent. of the required value would be sufficient. The simplest method of achieving this result is to switch the drum motor on and off at suitable intervals in the manner of a thermostat heater. The controlling element chosen was a photocell in conjunction with a source of constant illumination, arranged so that the cloud obscured some of the light from the lamp before reaching the photocell. The photocell current then depends on the optical density of the cloud. The limitations of this controlling element depend principally on the degree of constancy obtainable in the light source; experiment shows that a 75-watt lamp bulb driven by a 150-watt 'Advance' constant voltage transformer exhibits maximum fluctuations of about ± 3 per cent. in candle-power with normal A.C. supply mains fluctuations. The equivalent change in cloud concentration to produce the same change at the photocell is approximately ± 3 per cent., and so for the differential cloud concentration to be not greater than ± 5 per cent. the drum motor should be switched with about 2 per cent. change in illumination of the photocell. In practice this necessitates considerable amplification between the photocell and the relay controlling the drum motor. These considerations led to the design of the photocell relay described in the next section.

The Regulator. The optical system of the regulator is very simple, consisting only of a 75-watt lamp



V_1 = MADZA P.E.8.
V_2 = OSRAM H.63. OR Am. 6F5G.
V_3 = OSRAM GTIC
S_1 = SUNVIC VACUUM SWITCH.
T.D.S. = THERMAL DELAY SWITCH (BULGIN).
T_1 = 'ADVANCE' CONSTANT VOLTAGE TRANSFORMER 230 V 150 WATT.
T_2 = 0/230. 4V. 1·5A. AND 6·3V. 1·5A.
L. = 75 WATT. 230V. LAMP BULB.

R_1 = 10 MΩ.
R_2 = 50,000Ω.
R_3 = 1 MΩ.
R_4 = 20,000Ω.
R_5 = 5,000 Ω.
R_6 = 250.
R_7 = 6,000Ω.
R_8 = 0·25 MΩ.
R_9 = 0·25 MΩ.

R_{10} = 2215Ω.
C_1 = 0·1 μF.
C_2 = 1·0 μF.

FIG. 3.—The Regulator.

bulb, a portion of whose filament is focused with a single condenser lens on to the photocell, about 1½ metres distant. The circuit diagram of the complete regulator is shown in fig. 3. T_1 is an 'Advance' 150-watt constant voltage transformer of 230 volts output, V_1 is the photocell, V_2 the amplifier, V_3 a thyraton, and S_1 a hot wire vacuum relay whose contacts control the drum motor through the starting switch S_2. The operation of the circuit can best be described by assuming some light to fall on V_1, and considering the state of affairs at successive A.C. half-cycles. Take first the half-cycle when B is positive and A negative. V_1 passes current producing a voltage drop across R_1 so that the top plate of C_1 is negative with respect to the bottom plate. The time constant R_1C_1 is long compared with 1/50th second, so that the voltage across C_1 remains practically constant over one cycle.

Now consider the next half-cycle when A is positive with respect to B. V_2 now conducts, and its grid bias is controlled from three sources, and hence its anode current depends on three factors. Negative A.C. bias is derived from R_7, R_6; positive A.C. bias is derived from R_3, $(R_2 + R_4 + R_5)$, and this is variable; negative D.C. bias is derived from R_1C_1 controlled by V_1 as described above. The two A.C. biases partially counter-balance one another and are used so that R_1 may be made large to obtain a large voltage output from V_1. The anode current of V_2 produces a voltage drop across R_8 charging C_2 with the polarity shown in fig. 3, and the time constant of R_8C_2 is again large compared with 1/50th second.

Now consider the next half-cycle when B is again positive with respect to A. V_3 now conducts over part of the half-cycle—as controlled by the voltage across C_2—and the resulting anode current will flow through the hot wire control of S_1. Obviously at some value of anode current through V_3 the switch S_1 will close, thus turning the drum motor on, and this point is controlled by the grid bias of V_2. Thus by adjustment of R_4 and R_5, 'fine' and 'coarse' respectively, the additional D.C. bias required from V_1 to close S_1 can be varied, and hence the cloud concentration at which S_1 opens and closes can be varied at will. In other words the cloud concentration can be varied by adjustment of R_4 and/or R_5 after which it will be stabilized at the new concentration without further adjustment of the apparatus.

On striking, V_3 takes appreciable grid current which tends to charge C_2 with polarity the reverse of that shown in fig. 3. If $R_9 = 0$, then at the firing point the grid current of V_3 is more than the anode current of V_2, so that the latter current must be increased to a greater value than V_3's grid current before S_1 can open. In practice this means that there is some 'backlash,' i.e. the opening and closing illuminations are somewhat different. This 'backlash' or differential can be reduced or eliminated by making R_9 large. In practice it was found desirable to make R_9 variable so that the on/off period could be partially controlled. With no 'backlash' (i.e. R_9 very high) a change of illumination of about 0·2 per cent. was required to operate S_1, and since there were continual small fluctuations in the illumination, S_1 was opened and closed as often as once in two or three seconds, which was inconveniently frequent. The operator must strike a balance between too little 'backlash,'

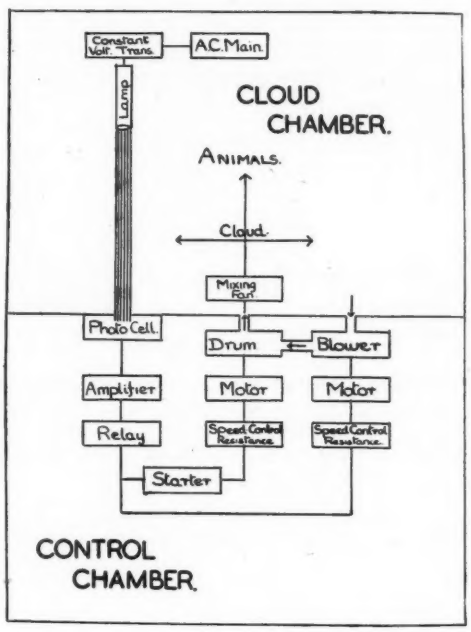
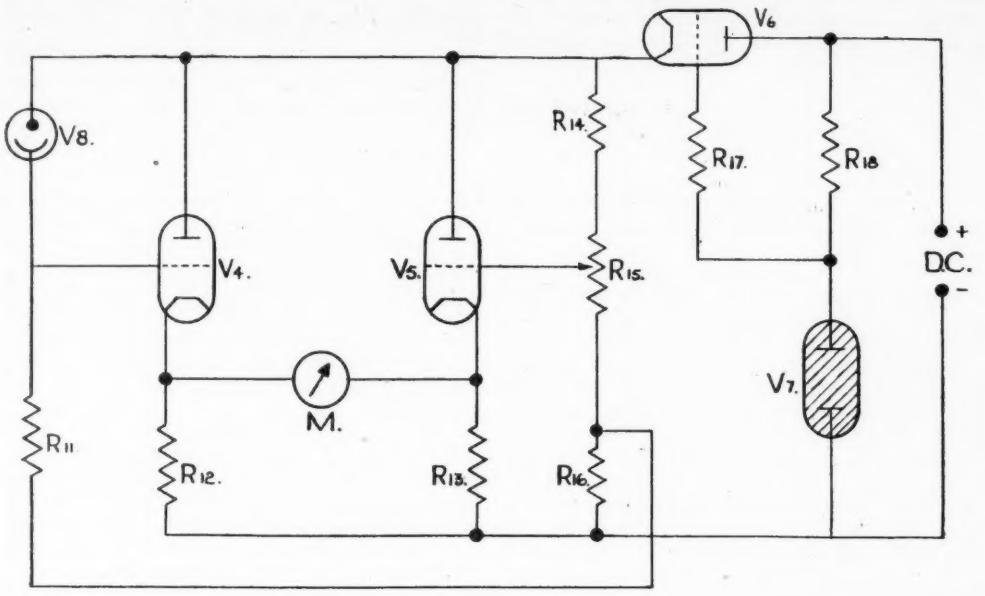


FIG. 4.—Block Diagram of Automatic Control.

unduly frequent operation, and too much poor regulation. In use this presents no difficulty.

One or two further points should be noted. The values of the resistances controlling the bias of V_2 will depend on the amplifier valve type chosen, the type of photocell used, and the illumination available. The values shown in fig. 3 were those used with the components shown and the illumination available at a cloud concentration of 250 mg. per cu. m. The thermal delay switch (T.D.S.) is used to allow V_3 to heat up for ¾ minute before applying anode potential. This is the manufacturer's recommendation for cathode preservation and T.D.S. also provides some protection against temporary supply failure. T_2 is a small transformer used to supply the heaters of V_2, V_3, and T.D.S.—two separate windings are required. R_{10} is adjusted to prevent overload of the hot wire section of S_1. It may be mentioned in passing that this regulator or photocell relay may be used for other purposes than that described herein, to operate stably with changes of 0·5 per cent. upwards. It would be of especial use when considerable illumination is available, heavy loads must be switched, and a time lag (due to S_1) of one or two seconds is not objectionable.



V4. = 6J7G. (TRIODE CONNECTED) Am.
V5. = 6J7G. (TRIODE CONNECTED) Am.
V6. = OSRAM H.63. OR Am. 6F5G.
V7. = MULLARD 7475.
V8. = MAZDA P.E.8.

R11. = 5 MΩ.
R12. = 0·03 MΩ.
R13. = 0·03 MΩ.
R14. = 0·075 MΩ.
R15. = 0·025 MΩ.
R16. = 0·015 MΩ.
R17. = 0·01 MΩ.
R18. = 0·03 MΩ.

FIG. 5.—Check Photometer.

The regulator has been in continual use for four months and has performed for about 700 hours. In that time one switch (S_1) has failed (it was in poor condition initially), one lamp has failed, one thyraton (V_3) has failed, and one resistance (probably initially faulty) has failed. Apart from these failures the only attention the regulator has required has been periodical cleaning of the lamp bulb. In use this became sooted over with charcoal, reducing the illumination, and hence the cloud concentration. No really satisfactory static self-cleaning device was developed, but something in the nature of a motor car wind-screen wiper would have been convenient. For the major portion of the running period the regulator was unattended.

Fig. 4 shows the general layout of the apparatus in diagram form. Details of the drum are shown in fig. 1, the starter switch in fig. 2, and the photocell amplifier and relay in fig. 3. The rest of the diagram is self-explanatory. A perspective view of the complete apparatus is shown in fig. 6. Fig. 7 shows a view of the cloud chamber, the animals being placed on wire netting shelves.

A Simple Check Photometer

It was thought desirable to have a check upon the absolute cloud concentration which might change due to the relative movement of the lamp and photocell on cleaning. In a more rigid optical arrangement this could perhaps have been avoided. To provide such a check a simple linear photometer was designed and constructed, the circuit diagram of which is shown in fig. 5. Additionally, the check photometer could be used to maintain the cloud with manual switching of the drum motor in the event of regulator breakdown.

V_8 is a photocell identical with V_1 and viewing the same lamp (fig. 3) through a portion of the cloud. Before starting the regulator each morning the meter M (50μA. 1500ω) was adjusted to zero with R_{15} (i.e. with no cloud). Then on production of cloud the illumination on V_8 fell, producing a

change of voltage at V_4's cathode, and hence an arbitrary reading on M. The cloud was adjusted by alteration (if required) of R_4/R_5 (fig. 3) until the regulator stabilized the cloud at a fixed but arbitrary reading of M. V_6 and V_7 provided a stabilized high tension voltage derived from the D.C. mains supply, which is further stabilized by the cathode follower connection of V_4 and V_5 so that variations in the supply input voltage are reduced by a factor of about 2000 before being applied to the meter.

Performance

Samples of the cloud were taken on filter paper and the concentration found by estimation of the trace element. In separate experiments the trace element/charcoal ratio was determined for (*a*) supply, (*b*) the sieved debris and (*c*) the sedimented cloud. The ratio was found to be sensibly constant within the limits of estimation error, showing that no alteration of the trace element/carbon ratio had taken place. In practice a sample was taken on each day's run as a check on the absolute concentration. In addition, pairs of samples were taken at hourly intervals on each of three days to check the performance of the regulator. A statistical analysis of the results showed that the required aim of ± 5 per cent. in the cloud concentration was achieved.

In addition to the regulator failures mentioned previously, in 700 hours one drum side was worn out and replaced by a spare, and the rubber of the rollers was renewed once. None of the failures was serious, the only fault causing a delay of more than a few minutes running time was the breakdown of the rubber rollers. Subsequently it was found that insulation tape was more durable, gave a better drive than rubber and was very much easier to replace. This fault caused a loss of one day (7 hours) in running time, and afforded an opportunity for a general overhaul. Care should be taken to prevent carbon from depositing on the electrical circuits and all apparatus should be earthed and circuits adequately supplied with fuses.

Results

The standard deviation σ_c of the chemical estimation and sampling errors was estimated by taking all samples in duplicate. The standard deviation of the overall variation of the results σ_r was calculated and hence the standard deviation of the regulator σ_R obtained. The calculated value of σ_R was 2·27. If the normal error curve is assumed, the apparatus

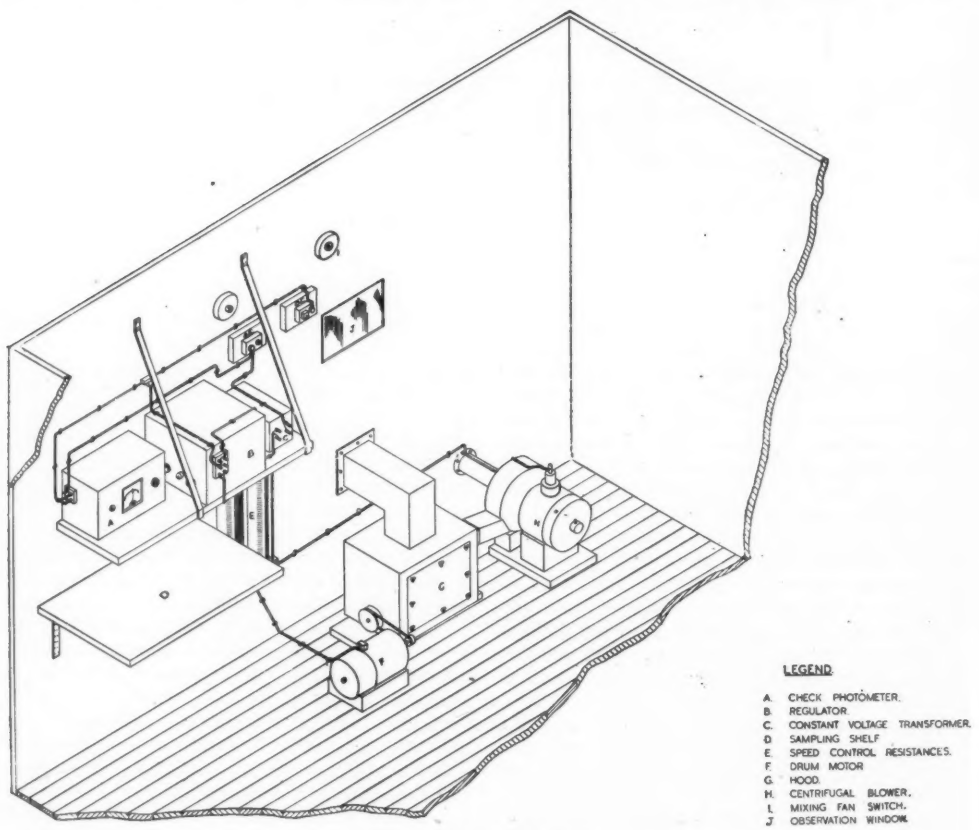


FIG. 6.—Control Chamber.

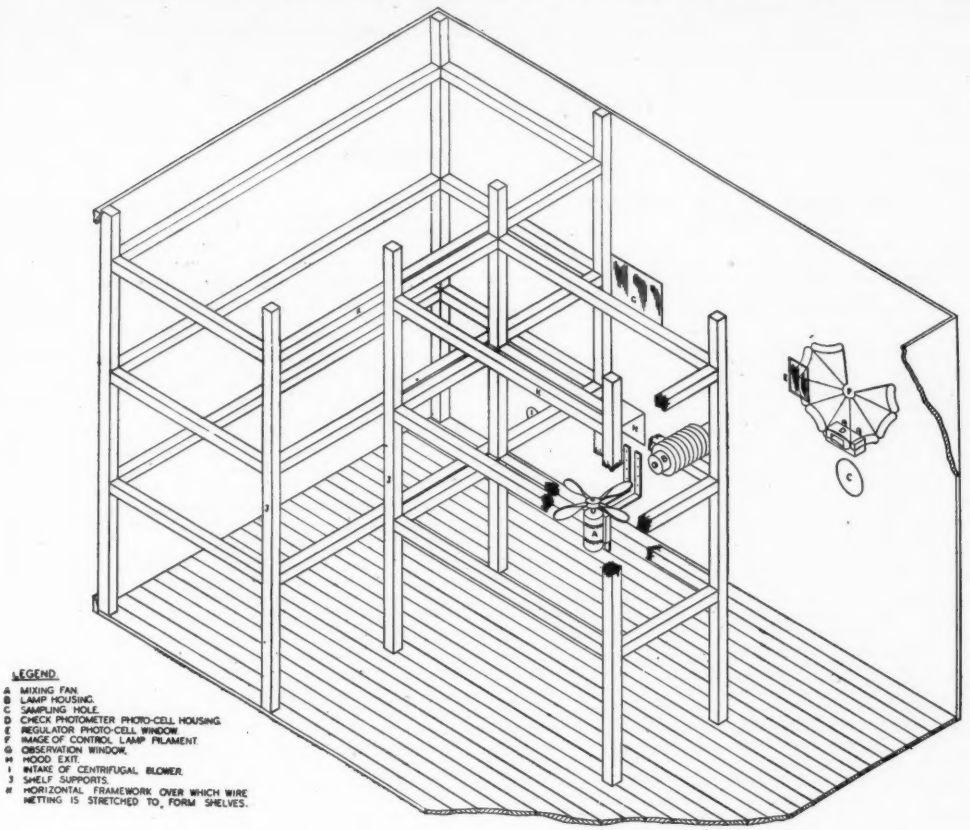

LEGEND.
A MIXING FAN.
B LAMP HOUSING.
C SAMPLING HOLE.
D CHECK PHOTOMETER PHOTO-CELL HOUSING.
E REGULATOR PHOTO-CELL WINDOW.
F IMAGE OF CONTROL LAMP FILAMENT.
G OBSERVATION WINDOW.
H HOOD EXIT.
I INTAKE OF CENTRIFUGAL BLOWER.
J SHELF SUPPORTS.
K HORIZONTAL FRAMEWORK OVER WHICH WIRE NETTING IS STRETCHED TO FORM SHELVES.

FIG. 7.—Cloud Chamber.

will be within ± 2·27 pet cent. of its adjusted value for 68 per cent. of the running time, and within ± 4·45 per cent. during 98 per cent. of the running time. As large departures from the mean (except for breakdowns) are impossible, the figure of ± 5 per cent. can be regarded as the outside limit of error.

Summary

An apparatus has been developed for the production of a carbon dust cloud in connection with physiological research on health hazard in charcoal filling factories. The cloud was produced by the attrition of charcoal in a rotating drum, and the maximum particle-size adjusted by elutriation. The drum was driven by a motor switched in the manner of a thermostat heater by a photoelectric relay controlled by the optical density of the cloud. The whole system provided a cloud of concentration 250 mg./m.^{3} ± 5 per cent. in a specially constructed chamber of 7·8 cu. m. for a period of 700 hours (7 hours/day). The apparatus was largely unattended except for a short daily period of standardization at the beginning of each run, and a second period of a few minutes at midday.

Acknowledgments

The authors express their thanks to Mr. J. Edwards, who designed and made the switch shown in fig. 2, and who assisted throughout the experiment. This article is published by kind permission of the Director-General of Scientific Research (Defence), Ministry of Supply.

BOOK REVIEWS

A TEXTBOOK OF THE PRACTICE OF MEDICINE

By various authors
Edited by Frederick W. Price. Seventh edition
(Oxford University Press. 1946. Pp. 2034. £2 2*s*.)

To have produced yet another new edition of 'Price's Medicine' during war is a wonderful achievement, especially in view of the great amount of new material included. The various authors have brought their sections up to date. The uses of penicillin are well covered. The thiourea and thiouracil treatments of hyperthyroidism, the latest diagnostic techniques in Addison's disease, thyroidectomy in the treatment of heart failure, the uses of the sulphonamides, the convulsant treatment of manic depressive states and the insulin treatment of schizophrenia are only a few instances of subjects brought up to date. In the section on blood diseases, always admirably concise and well ordered, some modifications have been made in line with recent developments; the treatment of haemorrhagic disease of the newborn by synthetic vitamin K is given, and there is an excellent summary of the pathology of erythroblastosis foetalis in the light of recent work on the Rh factor. Similarly, in the deficiency diseases section the most recent work is well collated, as well as clear and comprehensible. The section on endocrinology is particularly well done. Industrial toxicology has been given plenty of space, the metallic poisons, coal-tar derivatives and chlorinated hydrocarbons being dealt with in detail. The neurology section has been completely rewritten; as stated in the preface, the aim here has been at greater systematization, and this aim has been achieved. The presentation is clear and admirable in every way. Even though syndromes have been introduced which were omitted previously there is no feeling of over-concentration; indeed, as an example, the essay on aphasia is a model of lucidity. The textbook is an institution, and its worth increases with each new edition. Its sales are enormous. It is a pity, therefore, that some contributors still affect a medieval style. Some pages are still littered with fatal issues, impending dissolutions, and the *exhibition* of drugs including croton oil! One author deprecates the giving of milk to cases of vascular thrombosis because of the well known effect of calcium on the coagulation of blood. Another confides to us that he has always been disappointed by mistletoe. When readers as ignorant as the present reviewer have never known that any substance of value in therapeutics can be obtained from mistletoe, such a statement constitutes a mild psychic shock. The volume is still handsome, and the quality of the paper and print very good indeed. The only evidence of the difficulties encountered in war-time production is the slight increase in misprints; these are particularly noticeable in the section on diseases of the skin. As the editor states, the sale of this book abroad almost equals that in the United Kingdom. He rightly regards this as evidence of the international prestige of British medicine, but all will agree that medicine owes him, and his devoted collaborators, a great debt of gratitude for enhancing that prestige. D. H.

BRITISH AND FOREIGN OFFICIAL PUBLICATIONS

TOXICITY AND POTENTIAL DANGERS OF AEROSOLS AND RESIDUES FROM SUCH AEROSOLS CONTAINING THREE PER CENT. DDT

Various Authors. *U.S. Public Health Reports.* Supp. No. 183.

Two aerosols samples containing respectively 2·5 and 3·0 per cent. of DDT were first used, but later experiments were restricted to the latter since no appreciable differences were noted. Single exposure to high concentrations in a sealed chamber caused nasal irritation, restlessness, tremors and death in convulsions for some mice, particularly the younger ones. Rats were less affected and guinea-pigs less still; dogs only showed signs referable to the constituents of the aerosol itself. The DDT concentration was 26·4 to 32·9 mg. per litre and was maintained for 45 minutes.

Repeated daily exposures, for 45 minutes a day, over a period of 8 weeks with 33 mg. DDT per litre of air caused no injury to dogs beyond the symptoms of narcosis by the solvent, and exposure of monkeys, in the same conditions, for 22 weeks caused no signs of DDT intoxication or liver damage. When exposed three times daily the dogs died of pulmonary injury due to the solvents. The residue of the aerosols from which the propellant was omitted was easily absorbed through the skin of mice and 0·1 ml. was usually fatal by this route. Even applications of 0·012 ml. (2 mg. of DDT) were toxic and cumulative in under 3 weeks. Dogs died only after 12–21 weeks of daily repeated skin applications and, in contrast to the mice which died in convulsions, they died in severe depression, showing at autopsy jaundice and depletion of the fat depots only. The application of DDT here was massive.

Instillation of DDT aerosol into the conjunctival sac in rabbits caused irritation due to the solvents. Similarly local irritation followed subcutaneous injections, and only minor DDT intoxication resulted even after 200 mg. per Kg. were used.

Autopsy findings in general showed fatty degeneration of liver and kidney cells, and minor changes in the anterior horn cells of the mice. In those experiments the concentration of DDT put up was 3000–4500 times that usually used for entomological purposes, which is 0·004–0·001 mg. per litre. There is not likely to be danger to human beings from skin contamination under these conditions, but might occur from careless handling while filling aerosol bombs. *J. N. Agate.*

REPORT ON AN INVESTIGATION INTO DUST AND VENTILATION CONDITIONS IN THE COPPER MINES IN NORTHERN RHODESIA, WITH PARTICULAR REFERENCE TO SILICOSIS.

By J. de. V. Lambrechts, M.Sc.

(Government Press, Lusaka. 1945. Pp. 42. 2*s*. 6*d*.)

This work comprises a study of environmental and technical considerations in four copper mines and one

mine producing lead and zinc. The environmental study includes chemical analyses (*a*) of rock samples from working-places, (*b*) of dust samples from the sides and roof of airways, and (*c*) of airborne dust samples collected on filter paper. (The country rock was mainly quartzite, granite and sandstone.) X-ray diffraction equipment was not available, so that the free silica figures are possibly inaccurate, and in the case of airborne samples this estimation was not attempted. With this exception figures for total and free silica are given; while the free silica content of rock samples varied from 32·9 per cent. to 71·8 per cent., that in the settled-dust samples was from 22·6 per cent. to 53·6 per cent.

In the dust study the Kotzé konimeter was mainly used, and the Thermal Precipitator occasionally. It is stated that although the T.P. is an instrument of precision, its bulk and slow working-rate are drawbacks; the konimeter enabled a much larger number of working-places to be visited and this should more than compensate for the short sampling-time and any lack of accuracy in counting. Slides were treated by the standard (Ignition-Immersion-Ignition) treatment of the Union Mines Department. Good correlation was obtained between the konimeter and T.P., and a curve is given (fig. I)

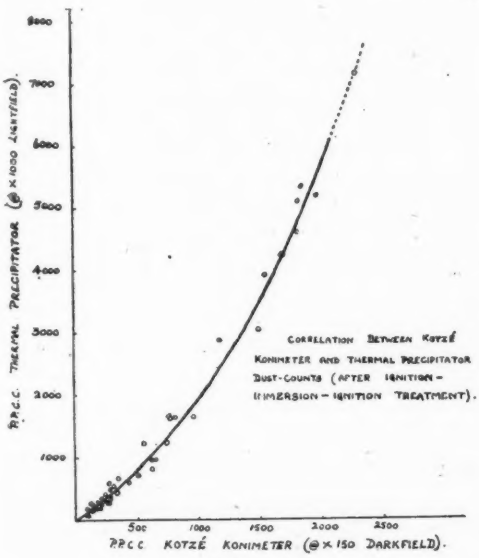


Fig. 1.

from which konimeter counts may be converted into approximate equivalent T.P. counts, provided that the particle-size grading is of the order found in this survey, and considered by the writer typical for wet or damp mines. It is indicated that correct interpretation of konimeter counts, especially high counts, is only possible by some such correlation, e.g. that a konimeter count of 1500 p.p.c.c. is not merely three times as bad as one of 500 p.p.c.c., it is about 4·4 times as bad.

Lambrechts deprecates the giving of average dust concentrations for a mine because (*a*) the averaging of very high and very low figures is arithmetically unsound; (*b*) to include figures for the safe places, the majority, with a few unsafe ones tends to mask the latter, though they may be causing most of the silicosis. The Standard of Permissible Dustiness suggested is a *maximum* of 600 particles per c.c. (konimeter).

It is stated that the chief dust-risks in order of importance, arise from:

(I) Primary Blasting. It is recognized that this should be dealt with on the usual lines, i.e. primary blasting only at the end of shift, followed by thorough ventilation to exhaust dust, and an adequate re-entry time.

(II) Secondary blasting, which in Rhodesia goes on intermittently throughout shifts, to break up very large pieces of rock to a size convenient for handling. This should be dealt with by specialized ventilation and use of water-blasts.

(III) Rock drilling. Axial waterfeed pneumatic percussion-drills are almost solely used, and a number of important observations on the use of fronthead-vented drills is given, thus (Table I).

TABLE I

	Rockdrill	Konimeter average p.p.c.c.	Remarks
Rhodesian Mines	Non-vented	1210	25 samples from non-ventilated ends.
Witwaters-rand	Vented	300	About 200 tests.

Vented drills reduce to a minimum the blowing of air as well as water down the channel in the drillsteel; a new non-vented drill may create ten times the dust that a new vented drill does; a vented drill without auxiliary ventilation produces about the same dust that a non-vented drill does with 1700 cu. ft./m. of ventilating air. (N.B. In New York State a statute governs the amount of ventilation to be supplied when rock-drilling; this amount depends on the relative rates of water and air flow through the drillsteel—the more air through the steel, the more ventilation required.) The writer suggests that ' an adequate flow of water ' through the steel should be defined as, at a water-pressure of 15 lbs. per square inch for vented, and 30 lbs. for non-vented drills.

Vertical-drilling stopers are deliberately designed to pass air up the drillsteel with the water, and have the additional disadvantage that they atomize the falling sludge-water from drill-holes, giving konimeter counts of 1500–5000 p.p.c.c. (3500–20,000 p.p.c.c. T.P.). The ' impact dust ' from these machines can be minimized by fronthead venting, and the ' atomized-sludge dust ' by directing the main exhaust port forward instead of backward (thus blowing the sludge away before it encounters the highest-velocity air at the port). Tables are given showing that the introduction of fronthead venting and changing the direction of the main exhaust port have resulted in 60–70 per cent. improvement.

(IV) Ore-transporting (mechanical-scrapers, hoppers, shaft loading boxes). These should be provided with adequate exhaust ventilation.

Ventilation. Fans are used; it is suggested that (*a*) the total air-circulation in the mine and (*b*) the rate of air-movement in each working-place, should be ascertained; and that dust-sampling results should decide whether ventilation is adequate.

Thermal Conditions. The relative humidity in working places is generally over 95 per cent. Curves are given showing rise of temperature with increase of depth. Attention is drawn to the conflict between the tendency to use water freely to minimize dust; and to use it sparingly to avoid humid conditions. No solution of the difficulty is offered.

Work in Surface Operations is considered (it is of interest that sidero-silicosis has occurred in drillsteel sharpeners on the Rand); and a section containing proposals for further legislation concludes this extremely interesting and instructive report.

L. W. Hale.

ABSTRACTS

Control of the Lead Poisoning Hazard in Can Manufacturing. I. The Bodymaking Machine (Side-seamer). EISENBUD, M., and MCEWAN, J. W. (1945). *J. Industr. Hyg.*, **27**, 284.

In almost all can manufacturing plants there is a bodymaking machine in which the side seam of the can is soldered. After the can has been formed, it passes over a solder bath about four feet long. The solder is applied by a roller, following which the soldered seam is heated by a burner. The cans then pass over a cloth wiping wheel which removes the excess of solder, and a blast of air is applied for cooling. The solder consists of 98 per cent. lead and 2 per cent. tin, and in order to investigate the amount of lead fume passing into the air at various points, a can line isolated from other can lines was studied. Samples of air were collected at the breathing zones by M.S.A. Electrostatic Precipitator, and analysed for lead by the dithiazone method. The data obtained showed that contamination of the air from the (well ventilated) solder bath is of minor importance compared with that from the burner, wiper and cooling blower; e.g. 10 cubic metres of air from these latter sources might contain 28–49 mg. of lead, as compared with 0·3–4·5 mg. from the former. When the wiping wheel was enclosed and ventilated by an exhaust, the air at all the points tested contained only 0·4–1·1 mg. of lead, or less than the permissible maximum of 1·5 mg. Samples of air taken at the same time from a room adjacent to the test room contained 2·9 mg. of lead. *H. M. Vernon.*

Studies of the Effects of Ammonium Picrate on Man and certain Experimental Animals. SUNDERMAN, F. W., WEIDMAN, F. D., and BATSON, O. V. (1945). *J. Industr. Hyg.*, **27**, 241.

A group of 71 individuals working with explosives containing ammonium picrate were kept under observation for 1–15 months. The ' milling ' and ' preforming ' groups were exposed to the greatest amount of picrate dust, and the picrate collected on the filter paper inserts in the respirators of two workers during a 6-hour milling operation yielded 52 and 156 mg. of picrate respectively. Seven of the workers developed dermatitis which was thought to be due to the picrate, but this may not have been the sole agent responsible because other components were occasionally present. The cutaneous lesions appeared usually on the dorsum of the hands, on the anterior surface of the forearms, and at the end of the elbows. They consisted mostly of erythematous patches containing papules and vesicles. Five of the seven individuals were able to return to work without developing further lesions, but they were urged to wear rubber gloves. The data obtained seemed to show that the persons least exposed were the most likely to develop dermatitis, and it was concluded that the picrate does not act as a primary irritant, but that sensitization to it may develop. The workers did not show systemic poisoning, but there was some staining of skin and hair.

When rabbits and guinea-pigs were kept for periods up to 12 months in the buildings where milling and preforming were carried on, over half of them died, and brown granular deposits, presumably picrate, were found in lungs, liver, heart muscle and kidney.
H. M. Vernon.

Observations on the Fujiwara Reaction as a Test for Chlorinated Hydrocarbons. WEBB, F. J., KAY, K. K., and NICHOL, W. E. (1945). *J. Industr. Hyg.*, **27**, 249.

Fujiwara discovered that when sodium hydroxide and chloroform are heated together, a red colour develops. The test is not specific for chloroform and has been used for the colourimetric determination of various chlorinated hydrocarbons. In the present investigation the influence of several factors on the estimation of carbon tetrachloride has been studied, and it was found that the test allowed the detection of the tetrachloride in concentrations as low as 2μg per ml. when it was dissolved in acetone. Also chloroform in concentrations as low as 1·3μg per ml. could be detected when dioxane was used as a solvent. Employing a fixed concentration of sodium hydroxide and using acetone as a solvent, the minimum detectable amount of a number of organic halides was estimated. Also the optimum concentration of sodium hydroxide for maximum colour production was investigated. *H. M. Vernon.*

Animal Studies on the Toxicity of Inhaled Antimony Trioxide. DERNEHL, C. U., NAU, C. A., and SWEETS, H. H. (1945). *J. Industr. Hyg.*, **27**, 256.

The pathological effects of the inhalation of antimony trioxide were studied in guinea-pigs. The animals were at first exposed in the experimental chamber for 2 hours daily, and after 3 weeks, for 3 hours daily. The average dust concentration in the chamber was 45 mg. of antimony trioxide per cubic metre of air, as determined gravimetrically by means of an electrostatic precipitator. It was computed that the guinea-pigs retained 1·6 mg. of trioxide daily, and that the 24 guinea-pigs employed inhaled from 13 to 424 mg. All the animals showed extensive pneumonitis, and 11 out of the 15 animals inhaling 77 mg. or more of the trioxide showed fatty degeneration of the liver. The white cells of the blood decreased, and so did the polymorphonuclear leucocytes and eosinophiles, but the lymphocytes showed a relative increase. No pathological changes were demonstrable in the heart or kidneys. *H. M. Vernon.*

Discussion on the Clinical Implications of the Disabled Persons (Employment) Act, 1944. BALME, H., STEWART, D., GRIFFITHS, H. E., AGATE, J., and WATSON-JONES, R. (1946). *Proc. roy. Soc. Med.*, **39**, 152.

Balme opened the discussion by saying that the Disabled Persons (Employment) Act has not yet received the attention which it merits from the medical profession who, with the hospital services, will be so directly involved.

In general terms, the purposes of the Act may be summarized as follows: First, it establishes the important principle that disability of any shape or form—whether congenital or acquired, medical or surgical, general or local—constitutes a claim upon the State for assistance, and that such assistance should not merely be expressed in terms of financial allowances or pensions, but rather in measures calculated to restore physical functions, wherever possible, and reinstate the disabled person in some suitable form of gainful occupation. Secondly, it makes provision for the vocational training and industrial resettlement of those who, subsequent to hospital treatment for injury or sickness, need to learn a new profession, trade or occupation before being fit to resume employment. Thirdly, it assists disabled persons to secure work by means of a requirement on employers to engage a stated proportion of men or women so handicapped. Fourthly, it provided employment under sheltered conditions of those who are severely disabled, but are able to do regular paid work, provided that they do not have to stand up to the working conditions of competitive employment.

A register is to be compiled of all persons substantially handicapped by reason of disability, and this will not only be a record of disabled persons for whom suitable jobs must, if possible, be found, but will also be a register of the failure of our hospital services to prevent residual disability. The particular directions in which medical assistance is essential to the successful operation of this Act, may be summed up in the phrases: Prevention of residual disability; assessment of loss of functional capacity; and periodic follow-up and review.

Certain well-established principles have emerged from experience of medical rehabilitation during the war: rehabilitation should be part of treatment prescribed by the physician or surgeon in charge of the case and not something secondary commenced and carried out during convalescence. The same principles regarding rehabilitation should apply to all forms of medical and surgical disability including a variety of chest complaints, to loss of abdominal muscular tone, arthritis, psychoneurosis and diseases of the central nervous system. Responsibility for all forms of rehabilitation treatment should be delegated to a single medical officer who should be responsible for the training and general supervision of the various ancillary workers. A carefully thought-out programme should be laid down for each type of medical and surgical disability, combining the use of physiotherapy and active exercises, remedial games and appropriate occupational handicrafts, alternating with prescribed periods of rest, relaxation and mental entertainment. The programme must always be related to the kind of work to which the patient hopes to return. The initial stages of rehabilitation necessarily take place in the wards and out-patient department, but the later stages are best carried out away from the hospital atmosphere.

Experience gained during the war has proved the value of physical reconditioning, selected occupational handicrafts and group games as adjuncts to expert psychiatric treatment for patients suffering from psychoneurotic disorders.' The almoner or welfare worker can render important service, since in large numbers of cases of illness or injury a psychological disturbance is present.

The hospital services must provide scientific guidance for the assessment of functional capacity and work in close co-operation with the Disablement Rehabilitation Officer, who has the responsibility of finding suitable work. The hospital which actually treated the patient should take the initiative in following up his case and arranging a review if this appears to be necessary.

Successful implementation of the Disabled Persons (Employment) Act will require the setting up of a comprehensive system of hospital rehabilitation throughout the country, providing four types of centres:

(*a*) An efficient physiotherapy department at every hospital for in-patients.

(*b*) A large out-patient rehabilitation centre (or centres) in every town or district.

(*c*) Residential rehabilitation centres outside the town.

(*d*) Special rehabilitation centres, scattered throughout the country, for the rehabilitation of particular disabilities.

The carrying out of such a programme will require a great expansion of trained staff and the inclusion of regular instruction in rehabilitation in the medical curriculum.

Stewart thought that occupations 'conducive to restoration of fitness' can be provided on a small scale in rehabilitation centres, but more important facilities can be provided in normal industry and in special rehabilitation shops and factories. The development of industrial rehabilitation must be accompanied by medical supervision at the place of work, that is, by a comprehensive industrial health service.

With good treatment the need for special rehabilitation measures decreases, but in the future, hospital authorities may have to consider the possibility of appointing their own industrial liaison officers who would not only provide a link with the patient's employer, but advise the surgeon concerning job-allocation. No matter how efficient the hospital, how good the treatment, and how much contact the social service or almoner's department has with the factory, there can be no effective return to work, and therefore, no full rehabilitation and implementation of the Act, without the co-operation of the employer. The employer's contribution must include promises of continued employment or alternative work scheme for retraining and rehabilitation workshops in the factory and assistance on matters arising under the Workmen's Compensation Acts. It should be a primary responsibility of personnel management, under the present system, to play a part in the supervision of the return to work of the sick or injured worker. The finding of light or alternative work is a matter of considerable difficulty, and often there is no adequate medical control. Medical supervision of return to work is of great importance and is a main reason for the extension of medical services to cover the whole of industry. The industrial medical officer should obtain accurate information of the incidence of sickness and accidents and of the progress of all sick and injured workers, and also compile an analysis of jobs and endeavour to establish special workshops in the factory. An account is given of a special rehabilitation shop set up in a large engineering works for injured employees who normally would not be at work, but would be undergoing treatment. During the 2½ years of its existence the shop employed over 500 injured workers and excellent results were obtained.

Griffiths pointed out that no mention has been made as to what the Act would cost industry if rehabilitation in its fullest sense were not achieved, and possibly 1½ million people would have to be found selected and unproductive jobs. The rehabilitation workshop has a greater educational than a curative value and the majority of cases will do better on alternative work.

Agate said if between 1 and 2 million disabled persons are going to register, notice must be taken of the Ministry of Labour's own estimate that of these, 25 per cent. have surgical disabilities, 50 per cent. have medical disabilities, and 25 per cent. are neurotic. The medical and neurotic cases can only be improved a little by treatment, and the Disablement Rehabilitation Officers are failing to place such cases in the right jobs. In many medical disabilities dyspnoea on exertion is the main handicap and determines the kind of work a man can do. Cardiac cases prove hard to place. A comprehensive job analysis for all occupations in the country is needed, but a possible alternative is for the doctor who knows the case of a disabled man to prohibit specifically certain physical actions, such as lifting weights of more than 20 lb. The new Ministry Form 'D.P.1' has this as its basis and is very helpful.

Watson-Jones thought that in most cases the resettlement of disabled persons called for very skilled medical experience coupled with a knowledge of industry. Disablement Rehabilitation Officers should be directed by local medical boards and their activities co-ordinated by regional medical consultants. Doctors should so influence their patients that a complete record was available of every individual who might have difficulty in seeking or keeping employment. If this was not done one of the greatest medical and social reforms of this generation would be imperilled. *A. Thelwall Jones.*

Backache. MEYERDING, H. W., and FLASHMAN, F. L. (1946.) *J. Amer. med. Ass.*, **130**, 75.

In this paper the authors discuss defects of the isthmic zone of the lumbar vertebrae as a cause of backache. The isthmic zone is defined as 'that portion of a vertebra between the superior and inferior articular processes. This areas is a narrowed bridge of bone and furnishes the only true bony support of the vertebral column.' According to Chandler, defects of the vertebral isthmus might result from faulty ossification, but Meyerding favours injury at birth or trauma superimposed on a congenital lesion as being responsible for such defects. Clinically the condition presents as low back pain which

is aggravated by any strain on the back. There may be a previous history of trauma. Physical examination may not reveal any abnormality, but there may be muscle spasm, tenderness or limitation of movement. Diagnosis depends on the history and the x-ray examination. It may be difficult to see the defect in the antero-posterior radiograph, but it is better seen in the lateral view and even more readily in the 'three-quarter oblique' view. Numerous radiographs are reproduced in which the defects are illustrated.

Conservative treatment of the defects consists in rest, support to the back and in physiotherapy to strengthen the muscles of the back. If this fails fusion of the vertebrae by bone grafts is advocated. Seven illustrative case histories are given. *W. S. Tegner.*

Silicious Exposures in the Fire Brick Industry. I. Engineering Study. STALKER, W. W. (1945). *J. Industr. Hyg.*, **27**, 275.

The refractory brick industry in Kentucky consists of ten major plants with a total working population of about 2000. Two raw materials are used in the manufacture of fire brick: (1) raw plastic clay containing 18 per cent. of free silica, and (2) raw flint clay, containing 7 per cent. of silica. Analyses of the dust encountered in the various industrial operations ranged from 1 per cent. of free silicon in burnt brick dust to 95 per cent. in the mould or parting sand, and averaged 12 per cent. The most dangerous sources of sustained dust production are the dry pan mills, with an average concentration of 580 million particles per cubic foot, and the free silica count was calculated to be 53 million particles. The average free silica concentration when grinding plastic clay was calculated to be 102 million particles, and 60 million when grinding burnt brick, so the accepted standard limit of 5 million particles is enormously exceeded. By enclosing the mill almost completely the average dust count was reduced from 108 to 10 million particles per cubic foot. The screen mills also produced much dust, and it was proved that this could be greatly reduced by their complete enclosure. The median particle size of all atmospheric dust collected in the industry was found to be under 3 microns, and was of the same general order as other industrial dusts. In most cases 74 per cent. or more of the dust was less than 3 microns and 90 per cent. or more was less than 5 microns. *H. M. Vernon.*

Silicious Exposures in the Fire Brick Industry. II. Roentgenologic Study. RITTER, W. L., and BOVARD, P. G. (1945). *J. Industr. Hyg.*, **27**, 283.

In order to determine the risk of silicosis to workers in the fire brick industry, x-ray exposures (17″ × 14″) were made on 87 per cent. of the men employed in seven major plants in Kentucky, or to 876 workers in all. On an average 11·0 per cent. of them showed pneumoconiosis, or from 6·5 to 18·8 per cent. in the individual plants. The fibrosis observed was uniform and finely granular, and seldom showed any tendency to form discrete nodules. The men had been employed in the industry for 9 to 47 years, or an average of 25 years. Two of the men—employed in dry pan mills—showed conglomerate silicosis, and 26 out of the 876 examined showed evidence of tuberculosis. The tuberculosis frequency was twice that found by the Tuberculosis Control Division of the State Board of Health during their routine mass surveys of the area.
H. M. Vernon.

Chronic Fatigue. ABRAHAMS, SIR ADOLPHE (1945). *Lancet*, **2**, 421.

A short paper in which chronic fatigue is evaluated as a symptom in cases where organic disease is absent. Manual workers seldom complain of it. 'Auto-intoxication' has been a popular explanation and has led to a spate of ingenious advertising. The psychosomatic approach might suggest that there is a relationship between the psychological accompaniments of fatigue and such a factor as hypoglycaemia. But the relationship is not exact. The professional classes often live irregularly and are out of training, expending too much energy on unprofitable mental activities, for which 'overwork' is later blamed. If work is congenial, there can hardly be an excess of it, and a complaint of overwork is not a cause but an indication of mental ill-health. Cabot's diagrams are shown, in which a properly balanced individual is compared with others in which there is imbalance from an excess of one or more of the four essentials, work, play, love and worship.
J. N. Agate.

The Aetiology of Silicosis. HALE, L. W. (1945). *Brit. J. Tuberc.*, **39**, 61.

Silicosis may be the earliest of occupational diseases. 5,000 B.C. neolithic man sank shafts into the earth from the bottom of which levels were excavated for getting the best workable flints as in Grimes Graves in Norfolk. Catarrhal infections such as chronic sinusitis, and repeated bronchial infections which temporarily or permanently destroy cilia predispose to silicosis. Dust which has entered the bronchioles and alveoli is no longer in contact with ciliated epithelium and depends on pulmonary phagocytes and the lymph vessels for its removal. Any inflammatory disease which involves the alveolar wall, lymph vessels or nodes, and produces scarring in these delicate structures will impair their function of carrying foreign matter and thus predispose to silicosis. It is in the phagocyte that the specific effect of silica is first seen. The cell enlarges, the cytoplasm increases, lipoid droplets are distinguishable in the cytoplasm, and later the nucleus may divide repeatedly, resulting in the formation of a typical Langhans giant cell. Some of the phagocytes in passing through these stages appear to become 'mummified' and their nuclear structure ceases to take stains, but the cell envelope may not disintegrate for a long time and the silica particles remain within it. The essential feature of the silicotic process is an excessive fibrosis in the intrapulmonary lymphoid aggregations, contrasting with the insignificant fibrosis of benign pneumoconiosis, together with a fibrosis of the perilymphatic connective tissue and a hyaline modification of the fibrous tissue due to the specific effect of silica. The nodules are tough, hard and contain gritty foci; they impede the flow of lymph, there is a zone of black surrounding them caused by carbon particles trapped amongst the fibrous tissue. The great majority of particles found in the alveoli measure less than 5μ and for this reason dust concentrations in atmosphere should be expressed as number of particles per cubic metre. Silica is excreted in the urine but no controlled investigations of silica balance have yet been made. Pulmonary tuberculosis is among those respiratory conditions which predispose to silicosis, while silicosis tends to make a man more vulnerable to tuberculosis. Even in modern hygienic conditions 75 per cent. of silicotics die of pulmonary tuberculosis. *K. M. A. Perry.*

Silicosis. DAYMAN, H. (1945). *Amer. Rev. Tuberc.*, **52**, 449.

A clinical study of 116 patients with silicosis, 33 of whom came to necropsy. Fifteen cases of nodular silicosis, 15 cases of simple conglomerate silicosis, 77 cases complicated by manifest tuberculosis and 9 cases unclassified, comprised the group. Seventy-two were iron miners, 22 stone workers, 10 millers, 6 coal miners, 4 foundrymen and 2 sand-blasters. The occupational histories of the simple conglomerate group are of interest since these patients had sustained less total exposure to silica dust than had other patients in the series, and were more often additionally exposed to carbon dust. The pathogenesis of these lesions is not clear; the non-silicous constituents of the dust may have some influence. There is evidence to suggest that the action of an infectious agent, tubercle bacilli, is additionally required,

and that in these cases histological as well as bacteriological evidence of tuberculosis has become obliterated. Pulmonary tuberculosis is the most important immediate cause of disability in silicosis; it is slowly progressive and does not respond well either to sanatorium treatment or collapse therapy. Cor pulmorale is a frequent complication and right sided cardiac failure is a common cause of death. It occurred in 26 out of the 116 cases, while there were clinical signs of decompensation in 16 cases, manifested by venous congestion, enlargement of the liver, and dependent oedema. Dyspnoea became aggravated by the onset of myocardial failure. X-ray demonstration of right ventricular enlargement is an important aid to diagnosis. The electrocardiogram showed right axis deviation. Other complications include fracture of ribs from muscular violence, enlarged lymph glands draining the lungs, chest wall abscess and obstructions of the biliary tract resulting from pressure on the common bile duct by enlarged silico-tuberculous lymph nodes. *K. M. A. Perry.*

Pneumoconiosis in Radiator and Boiler Finishers. DUNNER, L., HERMON, R., and BAGNALL, D. J. T., (1945). *Brit. J. Radiol.*, **18,** 377.

Of thirteen radiator and boiler fettlers examined with X-rays ten showed undoubted evidence of changes attributable to their occupation. The castings were made in sand moulds with cores of sand and oil. They were cleaned with wire brushes causing a cloud of fine dust. The radiological picture was that of the fine mottling in the middle and lower lung fields met with in pneumoconiosis. Three of the men were short of breath and two felt ill and unfit for work. The cause of the radiographic appearance is discussed. The dust inhaled contained iron, carbon and silica, but any conclusion as to which substance caused the mottling was considered unjustified. A plea is made for further investigation as to whether the lung shadows are due to actual dust deposits in the lung or to reaction of the lung tissue to the dusts. If the cause of the shadows is due to silica the men are entitled to compensation. *M. H. Jupe.*

Negative Chest Findings in a Mineral Wool Industry. CARPENTER, J. L., and SPOLYAR, L. W. (1945). *J. Indiana State Med. Assoc.* **38,** 389.

'Mineral Wool' is a fibrous glass-like material composed of calcium and aluminium silicates, and includes rock wool, slag wool and glass wool, of which the first two are discussed. None contains more than a trace of free silica but samples show from 29·5 to 51·5 per cent. of combined silica. Dust counts showed from 12 to 36 million particles per cubic foot, and 76 per cent. of the workers studied have had more than from 10 to 14 years' exposure to it. The only constant X-ray findings were increased linear markings in 51 per cent. of the group, the majority being from 30 to 54 years old, but a control group had a similar incidence of increased linear markings. This paper does not bear out a suggestion that dust from mineral wool is potentially dangerous, though the exposure here would have been long enough to produce abnormal X-ray appearances. *J. N. Agate.*

A Health Survey of Pipe Covering Operations in Constructing Naval Vessels. FLEISCHER, W. E., VILES, F. J. Jr., GADE, R. L., and DRINKER, P. (1946). *J. Industr. Hyg.*, **28,** 9.

The pipe coverers in two Government Navy Yards and two Navy Contract Yards were examined for the presence of asbestosis, incurred in their occupation. The pipe covering frequently employed was amosite, a magnesium iron silicate, which has a low thermal conductivity, light weight and refractoriness. Much asbestos cloth and asbestos cement were likewise used. Pipe covering may be divided into seven different operations, and dust counts showed that layout and cutting, band saw cutting, cement mixing and installation on board ship were usually the most dusty. Chest X-ray investigations were made on 1074 men in all, of whom only three showed signs of asbestosis. Each of the men affected had worked on asbestos pipe covering in shipyards for more than 20 years. It is recommended that the operations mentioned should be equipped with exhaust ventilation in order to keep down the dust concentration. *H. M. Vernon.*

Disturbances of Function in Silicosis. MICHAUD, L. (1945). *Presse Méd.*, **39,** 517.

The writer describes a disturbing increase in silicosis in Switzerland during recent years in consequence of which greater measures of protection are now introduced. Cases were first recorded in 1894 and in 1916 Staub published his case notes and induced employers to give greater protection and suitable compensation. This was the first legislation of its kind in Europe. Many suspected cases were later notified but in 1942–43 two-thirds of the cases suspected in miners could not be verified. It is pointed out that one must distinguish between mere anatomical change, physical signs and disturbance of function. The first may be present without the second or third. Conversely, a miner may complain of sundry symptoms and believe his capacity to be much reduced from silicosis, yet careful examinations show no sign of this disease. Diagnosis must be based on radiography and on the history. Screening alone is inadequate and serial photographs are needed. He describes three stages:

1. Minute shadows of pin-head size scattered in the middle zones with increased striation and enlarged or accentuated hilar shadows.
2. Shadows up to the size of a pea which are so numerous as to give a granular or tapioca appearance.
3. Compact shadows, usually in the middle zones and perhaps spreading upwards, resembling growths and with atelectatic areas; signs of contraction, fibrosis and pleural thickening and adhesions.

The appearances described are not sufficient, by themselves, to permit a diagnosis of silicosis. One must also know the history. Many other conditions may be mistaken for silicosis. There is no typical relationship between the period of exposure and the extent of the X-ray changes. Clinical signs and symptoms seen in these cases include râles and rhonchi, prolonged expiration, cough with fremitus. In general, there is evidence of bronchitis. Fever is uncommon. Patients tire easily, lose weight and complain of much dyspnoea even in the early stages. It is dyspnoea in particular which should make one suspect silicosis. Tests with the spirometer are valuable. Important evidence may be gained by exercising the patient and noting his pulse and respirations before and after. In the medical clinic in Lausanne much attention has been given to estimating the functional efficiency of the patient. As already stated there is no parallelism between the radiological changes and the degree of incapacity. Only in 60 per cent. of cases was a reduction of vital capacity determined, and this held good in those in Stage 1 and in Stage 3. In the other 40 per cent. there was an increased capacity, in relation to the theoretical values found by multiplying the theoretical basal metabolic figure by 2·5. Furthermore the maximal vital capacities do not show any even reduction corresponding to the stages of the disease. The values obtained, taken in the resting subject, give little indication of the degree of effort of which he is capable. For this reason it is necessary to get spirometer readings during exercise. There is at Lausanne an ergometer with which one may measure exactly any given output of energy. The work done is expressed in watts.

The maximum physical effort of which the patient was capable during a period of ten minutes in a 'steady state' was estimated. A 'steady state' was when the respiratory exchanges were stabilized without any

deficient oxygenation of the arterial blood and with a recovery period at the end of three minutes. No attention was paid to the output of energy when the patient is driven to exhaustion. If the energy output in ten minutes was 140, 130, 120, 100, 80 and 60 watts there was some relation between these readings and the stages of the disease. Patients in the second stage of silicosis may easily reach an output of 140 watts while some in the first stage only reach 120. But patients in the third stage, if capable of reaching 100 or 80 watts, cannot keep it up for ten minutes and so exhaust their respiratory reserves. Most of them show a fall in oxygen in the arterial blood. Patients are also found in the second stage who can only work up to 100, 80 or 60 watts by exhausting their reserves. But one patient in the third stage and aged 53 was found to be capable of reaching 120 watts in a 'steady state'. Another patient in the third state, aged 38, with a maximum respiratory capacity of 1·60 litres, reached 140 watts.

These results emphasize the lack of parallelism between the functional impairment and the morbid changes. The morbid changes are due not only to the deposition of silica, but to the associated hyperaemia of the bronchi, oedema, exudation and spasm.

P. H. Rossier has shown that vital capacity and respiratory deficit are increased by injection of adrenalin, which causes relaxation of bronchial spasm. Similar changes are seen in exercise.

The increase of dead space in these patients is connected with hyperventilation. Many factors are involved in the reduction in the patients' performances. Attempts are being made to find out more of the consumption of oxygen during effort in patients with comparable diseases. If the partial tension of oxygen in the alveoli is not enough to convert most of the haemoglobin into oxyhaemoglobin then the consumption of oxygen will be highest when the patient breathes an atmosphere enriched with oxygen. The impaired action of the heart, due to myocardial changes, displacement of the mediastinum and changes in the capillaries from lesions of vasomotor nerves, will all affect the readings obtained. Many other factors are also involved. The effect of emotional state in these patients must not be forgotten, especially as so many of them have witnessed the distressing development of the disease in their comrades. Workmen who merely exhibit structural changes are not entitled to compensation. To obtain this there must be loss of functional efficiency. *G. C. Pether.*

Estimation of Disability in Silicosis. HARRON, J. W. G. (1945). *J. Indiana State Med. Ass.* **38,** 391.

A correct diagnosis of silicosis must first be made, based on a knowledge of the hazard involved in an individual's occupation; the differential diagnosis from welder's lung, etc., must be considered. Normal clinical methods of examination give some picture of the disability; bronchospasm brought on by forced ventilation may be an important cause of this disability. The observation of any dyspnoea during normal activities is important. Respiratory function tests should always be correlated with the clinical findings, the history and X-ray studies, and notice taken particularly of the degrees of dyspnoea induced and the recovery times. The same equipment should be used throughout and a control series worked out from the normal individuals in the locality. Possible respiratory function tests are (1) measurement of tidal, complemental, reserve and residual air, and the vital capacity, (2) the normal minute ventilation and normal tidal volume, (3) the maximal minute ventilation and maximal tidal volume, (4) the pulmonary reserve, and (5) exercise tests on the bicycle ergometer or treadmill ergometer, or by other means, during which the expired air is collected and exercise minute volume measured. The greater the dyspnoea the closer does this figure approach the maximum minute ventilation. There is no substitute for time and experience in estimating pulmonary disability. *J. N. Agate.*

Physical Capacity in Work. HARVEY, V. K., and LUONOGO, E. P. (1946). *Occupat. Med.*, **1,** 1.

The big problem of evaluating the physical and mental capacities of all candidates for jobs is of such great sociological and economic importance that it overshadows the problem of the industrial capacity of the disabled; the latter problem is only part of the whole problem. The future of industrial medicine is closely allied to its success in promoting physical and mental hygiene, nutritional guidance, physical training and control of fatigue, and in the aiding of management in the selection of employees for specific types of work. Placement involves (*a*) an analysis of the job for physical requirements, (*b*) evaluation of physical capacity, and (*c*) matching capacities with the requirements of the job. It is necessary to realize however, that one of the most characteristic things about human beings is their variability, both in health and disease; and the greatest damage inflicted on either disabled or able-bodied by poor selection and placement, is not physical but psychological and results in loss of confidence, which may lead to more serious trouble with economic implications.

5100 different jobs in the federal service were analysed and their suitability for various kinds of disabled persons assessed. 1323 were suitable for persons with amputation of the thigh, 1464 for those with foot amputations and 50 for those with double leg amputations. The placement potentialities were broad and it was found that the frequency and severity of accidents were lower than those of an able bodied group of similar age, experience and occupational characteristics. For those with orthopaedic deformities of the leg 1373 jobs were possible, of the foot 1471, of both feet 46. Apart from deformities of the hip these workers had the same accident experience as the able bodied. Jobs for those with deformities of the upper extremities numbered 834, but the safety records of this group were not as favourable either in frequency or severity of accidents as those of the able-bodied.

With regard to cardiac patients, these should avoid occupations involving sudden spurts with unduly slack intervals of rest; they should have regular hours and favourable conditions of work, avoiding extremes of air pressure and temperature. The patients' capacity for economizing his energy should be considered, and it should be remembered that a heavy strain on the lower extremities is more easily tolerated than on the upper. No person liable to vertigo, syncope or sudden death should climb ladders, work near dangerous machinery or at high altitudes. Dusts induce cough and bronchial congestion and therefore should particularly be avoided by those with mitral disease, but any environment likely to cause infection of the upper respiratory tract should be avoided. Any occupation likely to cause anxiety or emotional disturbance should also be avoided. 1268 jobs were found suitable for cardiacs, and these patients' efficiency and productivity compared favourably with that of normal persons. Their accident rates were slightly higher with respect to frequency but as good with respect to severity.

With regard to placement of those with pulmonary tuberculosis, the job must not overtax the working capacity of the individual, and the working conditions must not be such as to be conducive to reactivation of the disease; and such that the patient is not a danger to other employees. 1328 jobs were suitable. The patients were found to have the same productivity and efficiency as able-bodied persons, while their safety record was better. Their rate of absenteeism for sickness however, was high.

1269 jobs were found suitable for deaf workers whose productivity and efficiency was as good as the able-bodied, but psychological factors in individual cases may produce an accident rate higher than that of persons with normal hearing. 2179 jobs were suitable for those who are hard of hearing and their productivity and efficiency compares favourably with the able-bodied though again psychological problems in individual cases increases their accident rate. They can often hear better in noisy

occupations. 2080 jobs were suitable for those blind in one eye ; their performance was good but the accident rate was high; it is believed that this is due to fatigue. They must be given jobs which do not cause danger to others; and care must be taken to protect the remaining eye. Fifty-five jobs were suitable for the totally blind, and these people were found to have as good a performance as the normal. *K. M. A. Perry.*

Restoring the Industrially Injured. HANSON, S. L. (1945). *Connecticut State Med. J.*, **9**, 759.

The industrially injured generally receive excellent surgical and hospital care, but recovery frequently lags in the convalescent period, and some cases do not get back to work because of the absence of a good introduction to work activity or appropriate placement when physical handicap has resulted from the injury. Inactivity prolongs disability and it sometimes develops discouragement or bad mental attitudes and fixations. No facilities have existed, however, for giving a medically supervised after-care to industrial cases that would prevent the development of these fixations.

Great success was reported from the Royal Air Force, who were giving a medically supervised after-care treatment, in the form of physical therapy and exercise, starting almost immediately following surgical operations. For the purpose of providing a facility where doctors could send the industrially injured, a Rehabilitation Centre was opened in Boston in June, 1943. Physical therapy was given concurrently with occupational therapy consisting of prescribed work in shops and also recreational activities. Of the first ninety-nine cases admitted to this centre, all of which were serious injuries, over 70 per cent. were either fully or partially restored to work capacity.

There is a growing demand for such facilities which is a challenge to the medical profession and hospital services to provide them. *A. Thelwall Jones.*

Selective Placement of Workers. POOLE, F. E. (1945). *J. Michigan State Med. Soc.*, **44**, 1060.

The views of industrial physicians and labour managers differ: the former is concerned with the amount and type of work an employee can do, the latter with the employee's job and skill. The physician's opinion is based on a physical examination supplemented by laboratory examinations and X-rays. Of 150,000 workers examined in the last four years, 79–85 per cent. were fit for all work, 16–12 per cent. fit for limited work and 5–3 per cent. unfit for work because of infectious disease, mental disorder or serious organic disabilities. A limited employee must not be employed on work harmful to himself or create a hazard to others; limited employees are classified :—I. No hazardous machinery, II. No heavy lifting, III. Ground level work, IV. Avoid contact with or exposure to (specified agent), V. No extensive walking or standing, VI. Special and Miscellaneous (for example, must work in a noise-free area). Employees returning after long illness should be classified before starting work. The employee's mental attitude is all important: with the wrong outlook, minor disabilities may be greatly magnified, and on the other hand, an over-ambitious employee may do himself harm. An adequate follow-up is essential as in particular the employee's immediate supervisor must be educated by personal contact by the medical department. To achieve full use of human power, supervisors who are completely familiar with production and technical methods require detailed and prolonged instruction. Supervisors should be encouraged to refer problems to the medical department, and on transfer to a new job limited employees should be re-examined.

At the start of the scheme for employment of limited employees, the plant average was 10 per cent. and the highest percentage in any department 15 per cent. These figures have now been increased to 14 per cent. and 26 per cent. of limited employees. The success of the scheme allows the problem of the returning ex-service man to be faced with confidence especially if the word 're-employment' is used instead of 'rehabilitation.' It is estimated that 10 per cent. of ex-service men will have disabilities, though 80 per cent. of this number will be capable of returning to their pre-war job. Only 15 per cent. of the disabled (1·5 per cent. of all ex-service men) will have handicaps needing special consideration. Greater emphasis must be placed on the psychosomatic and psychological aspect, though to submit all ex-service men to a detailed psychological examination (including tests) on return to the factory would be unnecessary and unwise. The ex-service man should be encouraged to resume his place as a normal man, rather than as a 'problem child' but all who do not settle down should be seen.

Industry does not 'owe' anything to the limited employee; properly employed the limited employee is not a hindrance, and the granting of special privileges transfers the employment of 'limited employee' to the realm of charity, unjustified unless grants-in-aid are made by the Social Security Board or State Rehabilitation Commission. Quite apart from medical ethics, caution must be exercised in telling the management about the limited employee's disability in case over-caution by the supervisor reacts to the employee's detriment. Physical disabilities are easier to place, and when properly placed capable of doing a full job; but degenerative conditions present the greatest problem and, it has to be recognized, do result in increased risk to the employee and to industry. *T. A. Lloyd Davies.*

Rehabilitation of the Blind. FEIMAN, E. M. (1945). *Industr. Med.*, **14**, 649.

The author describes a very ingenious method by which the blind can be employed on precision work.

In manufacturing tapered roller bearings it is essential that the diameter of the various parts for anti-friction bearings be ground to within very fine dimensions of the specified sizes. Those parts falling over and under the specified size limit are detected and removed by means of an electronic device. To enable a blind operator to complete this very fine inspection with the same accuracy as a sighted worker, the Timken Roller Bearing Company has invented a sound detector which gives a high note indicating oversize, a middle note within the specified limits, and a low note indicating undersize. Thus it is possible for the blind to do inspection work within one twenty-five millionth of an inch, using their ears. This apparatus is available to any industry willing to employ the blind for inspection work without any rights or royalties.

Blind employees do sound tests which are the final inspection of the assembled bearings for smooth and noiseless operation and also file testing by feel for hardness of steel. It has been found that the blind can do about 80 per cent. as efficient work as the regular employee. *R. S. F. Schilling.*

Problems Arising from Michigan's New Compensation Law. RYAN, T. P. (1945). *J. Michigan State Med. Soc.*, **44**, 1056.

From 35 years ago, when the question was whether to adopt a compensation law (all states have except Mississippi) the problem to-day is how to broaden the law so as to include all workers and all types of industrial injuries. (Each state in U.S.A. has its own compensation law. In Michigan, Workmen's Compensation is administered by a commission and a lawyer describes its workings.) Only those workers whose employers are subject to the law are protected by it. Prior to 1943 the law was elective for all employers (40,000 employers being so subject) but since that date the law is compulsory for all employers of eight or more persons (over 50,000 employers being subject). The Unemployment Compensation Law is similarly compulsory for employers of eight or more persons, and under this law 18,000

employers are registered so that the majority of the 50,000 employers subject to the workmen's compensation law employ less than eight persons.

In 1937 scheduled occupational diseases were admitted for compensation but many anomalies occurred, for example, a grinder received compensation for pneumoconiosis but not a moulder working in the same room and exposed to the same hazard. The 1943 amendment removed the requirement that industrial injuries must be accidental to be compensatable, and general occupational disease was substituted for schedule disease. Compensation is paid both for injury due to a single event and not due to a single event, for example, disease. It may also be paid for aggravated disease. To receive compensation a disease must be personal and be 'due to causes and conditions which are characteristic of and peculiar to the business of the employer and which arise out of and in the course of the employment'. A dissenting judgment (of greater legal significance than the majority) is quoted in which a woman using the hands for manipulating bottles suffered from bursitis but because this type of work did not carry an increased risk of developing bursitis, she was not (in the opinion of the dissenting judge) entitled to compensation. A woman developing arthritis and partial subluxation of the proximal metacarpal phalangeal joint of both thumbs in the course of filing castings, was on this basis refused compensation by the commission, as the injury was not 'due to causes and conditions characteristic of and peculiar to the business of the employer'. This case has gone to appeal. In the author's opinion that is the criterion by which eligibility for compensation must be decided. Compensation is not intended to cover the chronic ailments arising in advancing years. Sometimes the division between natural illness and injury is vague, and distinction depends not on the nature of the disease but on circumstantial evidence, for example, a hernia in a man doing heavy work is probably occupational in origin but in a man doing light work probably arises naturally.

Consideration of these matters should be kept in the hands of competent persons of good judgment who know the difference between speculation and proof.

T. A. Lloyd Davies.

Industrial Accidents and New Legislation: Some Implications of the National Insurance (Industrial Injuries) Bill, 1945. STEWART, D. (1946). *Brit. med. J.*, **1**, 561.

In changing the basis of workmen's compensation to a contributory scheme, the National Insurance (Industrial Injuries) Bill, 1945, should remove many of the workman's grievances with the present system, and, it is important to note, change the work of the medical profession. The employee will contribute to the Industrial Injuries Fund by weekly stoppage of pay 4*d.* for men, 3*d.* for women, 2½*d.* for boys under 18 years and 2*d.* for girls under 18 years. The employer will pay similar amounts. The payments are additional to the contributions to the National Insurance Fund, and already workers have said they intend to use this compulsory levy in an argument for increased wages. The fund will be aided, if the Treasury think necessary, by an Exchequer contribution equal to one-fifth of the aggregate amount collected, making a total (with the Exchequer's payment) of £27½ million a year.

All persons under any contract of service or apprenticeship in Great Britain are insured (excluding the armed forces, police, certain workers not yet prescribed in other Crown employment, or under a public or local authority). Of the 13½ million men and 4½ million women classified as employees in the Social Insurance Report, 1944, approximately 10½ million will be insured for industrial injuries. In 1938, 460,000 persons received £6¾ million in compensation under the Workmen's Compensation Act, out of a population at risk of 7,800,000.

Benefits are not based on loss of earning capacity (which may be different in different persons with identical injuries) and the tendency to consider this particular section of the social insurance plan in isolation from the general scheme is unwise, and has raised industrial injury payments above the general level. No regard has been paid to the cost to the community, but even so the T.U.C. are pressing for an increase of the industrial injury benefits from 45*s.* to 55*s.* per week. The National Union of Mineworkers considers that the dangerous nature of their work warrants an increase of 50 per cent. increase in benefits. Differences in payment may retard treatment and rehabilitation, and in any case the nation cannot afford an incentive to remain out of productive work.

Under the new system, a claim for benefit involves five persons, the workman, employer, insurance officer, office clerk and doctor. Will this cause delay in payment, and will the introduction of government officials into an intimate health circumstance and a private matter between man and master cause difficulty? The scheme must be administered with great care for human decencies, and to do this many officials will need training.

Medical certification of fitness for work will be required in increasing amounts, and steps may be taken to make certification more accurate and based on a knowledge of job requirements. In the interest of the patient, rehabilitation services should become the responsibility of the new health service rather than the field of independent action of the Ministers of National Insurance, Labour, Fuel and Power, Pensions and Health. A national accident service is a logical outcome.

Medical boards, similar to or even the same as those now constituted by the Ministry of Pensions, will be needed, but if the disability is likely to be less than three months, and with the consent of the claimant, the Minister may refer the case to a 'single' medical practitioner who may be an industrial medical officer. One of the most important jobs of an industrial medical officer is to assess the fitness for work, but this should be done in consultation with the patient's private medical attendant. Because the financial success of the Bill depends on the prevention of accidents and disease, the industrial medical officer, both now and in any future industrial health service, will be called upon to contribute to research in industrial health. This is especially important, and the Act specifically empowers the Minister to promote research. But will the Industrial Health Research Board continue to be understaffed and inadequately housed, or will the Ministry set up its own research organisation? To conduct adequate enquiries into the cause of accidents a national health statistical department will be required. In addition to the Advisory Council provided for by the Act, it is suggested a medical advisory committee should be set up to advise the Minister on the many and varied medical problems (especially the assessment of prescribed industrial diseases) so that the influence of the medical profession may attain its full effect. *T. A. Lloyd Davies.*

Visual Discomfort in Eye-Workers due to Glasses. BERRY, G. L. (1945). *Oklahoma State Med. Assoc. J.*, **38**, 361.

More patients complain of visual discomfort than poor vision, so satisfactory refraction calls for comfortable vision as much as a normal visual acuity. Poor vision is never uncomfortable, even if it is inconvenient but active use of the eyes for near vision is uncomfortable whether for an emmetrope or a corrected myope. The causes of discomfort are manifold, and astigmatism is the most common; others are asynkinesis as in hypermetropes, where the amount of convergence required does not equal the amount of accommodation, excessive power of both accommodation and convergence which can in part be remedied by overcorrection of the refractive error, or low power of both accommodation and convergence which requires either orthoptics, fixed prisms incorporated in the patient's glasses, or a combination of the two methods. In measuring muscle balance the prism bar is more accurate than the Maddox Rod; it is less confusing to the subject and yields more information. The more common causes of uncomfortable vision, due

to the glasses themselves, are faulty measurement of interpupillary distance at the range at which the patient works, vertical imbalance induced by the correcting lenses, either when the lens grinding is at fault, when the patient looks down through two different powers of lens, or when there is no decentration to correct anatomical differences of height of the eyes. Some patients have an asymmetrical nasal bridge which again calls for decentration of the lenses. Cylinders used to correct astigmatism have in certain positions secondary prismatic effects or induce artificial cyclophoria which cause discomfort. Many cases of 'eye-strain' are due to low convergence with low accommodation, and these sufferers need bifocal lenses. Untreated weak convergence accounts for more discomfort than all the others together, and patients can sometimes be helped with thyroid extract and a little base-in prism effect. Poor accommodation may be due to ciliary weakness or spasm, and result in having no reserve for close work, in which case a little overcorrection will give some reserve, which ought to be one-third of the total accommodation. A lens grinder cannot produce satisfactory lenses unless the refraction is accurate and his instructions complete in every detail. *J. N. Agate.*

Psychiatry in Industry. CAMERON, D. E. (1945). *Canad. med. Assoc. J.*, **53**, 538.

The industrial psychiatrist has three main functions, teaching, clinical practice and research. Managers, foremen, doctors, nurses and employees must be taught the facts of human behaviour. The more a man knows about himself and others, the less frustration will he encounter; the more executives know of human motivation, the more efficient they will be. Teaching should emphasize that any job analysis must take into account whether the job can give satisfaction to the worker and to what kind of worker.

The importance of psychosomatic medicine is not yet realized; many are still ignorant of all the persuasive symptoms of tension, even those who can evaluate it in gastro-intestinal and cardiac disorders are ignorant of the nature of many cases of tinnitus, dizziness, blurred vision, postural kyphosis, pruritus and many dermatoses.

If we overload the man who is excessively keen on doing his job well, he will become ill and finally be lost to industry. Another cause of serious wastage of valuable employees is placing incompatible individuals together as when a highly gifted but emotionally insecure man is placed with an assertive, aggressive boss.

Psychiatric principles must be applied by the whole ndustrial team, as the surgeon with his asepsis, the housewife with her insistence on pasteurized milk, the engineer designing a drainage system are applying the principles of bacteriology. The specialist psychiatrist should assess the personality at intake into industry, be available for consultation when required and, by suitable employment recommendations, ensure that nobody is employed where he will certainly break down.

Research must discover what kind of jobs and situations produce excessive tension, in what kinds of persons serious degrees of tension will occur and how such tensions can be prevented and cured. *E. H. Larkin.*

Psychosomatic Medicine in Industry. ROSS, H. G. (1945). *J. Michigan State med. Soc.*, **44**, 1067.

If health is to be a positive conception, the patient must be considered as a whole in relation to his environment. To this study, the term psychosomatic medicine is applied. Medicine has become sectionalized, and in spite of many purely scientific advances in medical knowledge, a return to the practice of the art of medicine is the essential foundation for psychosomatic medicine. The status of an industrial physician as a specialist is the subject of controversy but the industrial physician needs to have a broad and broadening vision rather than the narrowing view of the specialist in other branches of medicine. Because of his opportunities of studying the patient's working and home environment, the industrial physician is in a favourable position to apply the principle of psychosomatic medicine. The sickness-prone worker may be of three types:—I. those of poor physique, of bad heredity, from a bad home or suffering from 'just bad luck'; II. those with many complaints for which no organic cause can be found but who are actually ill; III. those who suffer from an organic disease complicated by psychic factors. The two latter groups present the greatest problem to medicine, industry and the community.

Treatment consists of explaining to foremen that such persons' complaints are genuine, the relief of anxiety and emotional tension, the adjustment of housing, money and other worries. Those with recent emotional upsets respond better to treatment than those whose symptoms originate from childhood. Because so few psychiatrists are interested in industrial medicine, the industrial physician must, by exercise of common sense, apply the principle of psychiatry to these cases.

(To English readers this paper presents some confusion in terms. The first part of the paper is an admirable exposition of the need for the study of social medicine—to which the author applies the term psychosomatic medicine. The second part explains the psychosomatic origin and the need for psychiatric treatment for patients whose symptoms are not, in the true sense of the term, psychosomatic in origin, i.e. Group I in the author's classification.) *T. A. Lloyd Davies.*

An Industrial Mental Hygiene Programme for Federal Employees. CRONIN, J. W., SOLBY, B., and WILDER, W. S. (1945). *U.S. Publ. Hlth. Reps.*, **60**, 1323.

Only organizations employing large numbers of persons can create a fully staffed mental health unit, but smaller firms might combine for this purpose. This would need a uniform and scientific system of personnel management, though in small firms management is largely on a personal contact basis which often leads to a satisfactory job adjustment, loyalty and morale. As Industrial Mental Health Service is in its initial stage, centralized services would enable useful data to be collected and offer special service, such as psychometric testing to small firms.

Symptoms of maladjustment vary according to the war position: in times of urgency the human factor tended to be emphasized by management, and the individual worker found increased satisfaction in his job. Slackening of the war effort and the consequent insecurity in national and individual plans for the future lead to acute psychological conflicts and emotional disturbances especially in those who wished to be released from Government service.

In planning an industrial mental hygiene unit, administrators, personnel managers, and foremen, and even doctors require education in the need for such a unit. So long as psychiatry was based on instinct and interpersonal reactions it had little to offer industry. Industrial psychology stresses the importance of the job as a major factor in the happiness of the normal adult.

Most of the patients referred to the Mental Hygiene Unit set up in July, 1943, as part of the U.S. Public Health Service, showed poor job adjustment. As experience was gained, this was shown to be less related to difficulties arising outside work and more to the job itself. In fact, good job adjustment is necessary for the attainment of a full life. But, in arriving at a diagnosis, a full account of the patient's work, medical and social background is needed. Because of the specialization of skill required, and the recognition of his dependence on other members of the group, the industrial worker is likely to be more outspoken in his requirements than agricultural or clerical workers. The ease with which patients discuss their difficulties is surprising, but probably due to the fact that the psychiatrist assumes the role of technical expert helping to solve the patient's difficulties. Transference on an adult level rather than on an infantile level takes place between patient and psychiatrist. To ensure industrial mental health much educational effort will be required. Job placement,

adequate supervision and training are especially important. In particular, the foreman must expand his role from production expert to a leader of those for whose work he is responsible. *T. A. Lloyd Davies.*

Industrial Hygiene in New Zealand. DAVIDSON, J. M. (1946). *Industr. Med.*, **15**, 226.

Health risks of a toxic nature are relatively rare in New Zealand, but those that exist are important because so far they have frequently passed unrecognized. This country's system of social security aims at providing outside the factory facilities for medical treatment, but makes no provision for the supervision of the health of workers at work. But there are a few factories in which progressive managements have appointed part-time medical officers and nurses. Nevertheless, where a doctor has been appointed his duties are directed towards curative rather than preventive work. The standards of first-aid and of first-aid equipment are low, individual sealed sterile dressings are unknown, and frequently no records are kept of persons treated.

The practice of preventive medicine in industry will be a new field for New Zealand doctors and nurses, but it can only be applied to industry if industrialists appreciate the value of skilled medical supervision which means a great deal more than a glorified casualty service or having a doctor 'on call'. *R. S. F. Schilling.*

Industrial Medicine in the Philippine Islands. FELTON, J. S. (1946). *Industr. Med.*, **15**, 129.

Owners of commercial, industrial or agricultural establishments in the Philippines are obliged to give free emergency medical attendance to their employees. This applies not only to private industry but to all Government establishments. Specific instructions are laid down as to the types of medical facilities to be given; their scope is determined by the numbers employed. For thirty workers a stock of emergency medicines must be maintained under the charge of a nurse or other intelligent person. When the industrial population exceeds 400 persons, the owner must employ the services of a physician and provide a dispensary or an emergency hospital. The Bureau of Health has inspectors to see that the law is enforced.

Philippine industries include mining, lumber, sugar, rice, hemp, tobacco, textiles, rattan and leather. The author describes the sugar industry in detail. Although occupational health risks must exist in this industry, workers claim little knowledge of such illness. There has been no morbidity due to exposure to bagasse, but dermatitis due to phenolic sprays for cleaning occurred until steam was used as a cleaner. The plant medical service provides care for the worker and his family at a cost to the worker of 1 per cent. of his salary. Preplacement medical examinations are necessary by law, but there is no varying set of standards for jobs with different physical demands. All workers irrespective of their job must meet the same physical standards before they can work. The main contra-indications to employment are hypertension and tuberculosis. The latter disease is extant in the islands and its spread is feared by the people. Health education is very necessary because the average Filipino will not consult a doctor in the early stages of disease, but pamphlets have not been so successful as visiting nurses who can get into the provinces and *barrios*.

There is a Workmen's Compensation Act which provides adequately for the disabled.

The employer is required to pay all medical and surgical costs following injury or illness incurred by one of his employees. *R. S. F. Schilling.*

Industrial Health Activities of Government. TOWNSEND, J. G. (1945). *J. Indiana State Med. Assoc.*, **38**, 373.

The U.S. Department of Labour has Bureaux of Labour Statistics, Labour Standards, and others to supervise the employment of women and children. The U.S. Public Health Service incorporates an Industrial Hygiene Division and Industrial Hygiene Research laboratories. Since 1935 various occupational disease laws and the Social Security Act have been applying in practice the knowledge gained from previous research. Industrial hygiene started by considering traumatic injury at work and has evolved to concern itself with protecting every worker in industry and with ensuring the greatest possible physical and mental health for each. Forty-nine official industrial hygiene units are located in thirty-nine States.

The Industrial Hygiene Division of the U.S. Public Health Service is made up of (1) The Field Operations Section; (2) the Dermatoses Section; (3) The Medical Unit, which is consultative and undertakes epidemiological investigations; (4) the Engineering Unit; (5) the Dental Unit; (6) the Nursing Unit; (7) the Chemical Unit, and (8) the Statistical Unit. The Division working in co-operation with all manner of other interested bodies kept watch over working conditions in government munition plants, and disabilities and deaths from toxic exposure in making explosives were greatly reduced; it made a study of shipyard workers and the effects of welding; it undertook to protect the health of workers in 379 foundries and forges in twenty-seven States, and of lead smelters in twenty-two States: it studied conditions in chromate plants, and in aluminium reduction and asbestos textile industries. It concerned itself with nutrition of war workers and prevention of accidents and illness in a variety of industries.

Laboratory research was directed to the toxic properties of explosives and DDT, but in particular of non-inflammable battleship paints, and the ingredients of synthetic rubber. High altitude temperature and pressures, oxygen breathing equipment, oxygen-helium resuscitation methods, and tropical conditions were all studied.

The Industrial Hygiene Division has assisted work in the individual States with funds, trained personnel, laboratory facilities, loan of equipment, consultative opinion and published literature.

The main post-war difficulty is the shortage of trained industrial doctors. In 1939 the State of Indiana had only 10 per cent. of its workers supervised by a full-time industrial medical officer. Fundamental research is to be directed to adjustment of handicapped war veterans to industry, industrial dermatoses, health problems of air transport, hazards of atomic energy production, and the toxicity of new chemicals and plastics. *J. N. Agate.*

Industrial Dentistry. HEACOCK, L. D. (1945). *J. Indiana State Med. Assoc.*, **38**, 381.

Industrial dentistry lapsed between the World Wars but was resumed in the recent one. Minimal standards were laid down in 1941 by the American Dental Association. The Dental Unit of the Industrial Hygiene Division of the U.S. Public Health Service has assisted state health departments to correlate their activities and tackle industrial dental problems, and three of them employ full-time dental officers. Routine oral examination with X-ray can be done in the reasonable time of five or six minutes. Some industrial hazards are first noticed because of symptoms related to the mouth, as in the case of sore gums occurring as the first sign of poisoning in a plant using metallic mercury to make electric batteries. The industrial dentist needs to know the physical and toxic hazards involved, and must keep full records.

A five days' course was given in December, 1944, at Columbia University on subjects relating to industrial dentistry. The essential parts of an adequate service are (1) examination and diagnosis; (2) emergency treatment; (3) recording of findings; (4) dental health education, and in a small plant a local dentist could be 'on call' to give them, while in the larger, full or part-time services are the sounder proposition.

J. N. Agate.

Progress in Industrial Dermatology. PECK, S. M. (1945). *J. Indiana State Med. Assoc.*, **38**, 383.

Before the war the incidence of occupational dermatitis in the U.S.A. was one case in every hundred employed workers. In war industries the figure amongst new employees sometimes reached 50 per cent. The agents mainly responsible are petroleum products and greases, alkalis, and solvents. Full knowledge of industrial processes besides experience in dermatology is needed for accurate assessment of the causes of the dermatitis. Postgraduate schools could train industrial dermatologists; the U.S. Public Health Service has instituted concentrated lecture courses. Lectures are suggested for industrial nurses, safety men and the workers themselves. Research has been hampered by lack of interest: such problems as that of cutting-oil dermatitis need tackling. Large industries with full-time medical staffs can keep abreast of advances in dermatology, but the smaller ones are behindhand owing to lack of special list facilities.

J. N. Agate.

Dermatitis due to a Case-hardening Compound. WHITWELL, G. P. B. (1945). *Brit. J. derm. & syph.*, **57**, 169.

The case-hardening of steel consists essentially in bringing carbon into contact with the metal surface in such a state that the steel is made richer in carbon content. The process can be done in various ways, such as packing the metal round with charred leather, bones or charcoal, and heating in a furnace; or the metal may be suspended in a bath of molten sodium cyanide. Dermatitis should not occur from this latter process.

The dermatitis described is due to a combination of the two above processes, when organic carbonizers are mixed with chemicals. In one process wood charcoal, impregnated with 5 per cent. sodium carbonate, is used, and in another, wood charcoal impregnated with barium carbonate. Much soiling of the exposed skin occurs and severe eczematoid dermatitis is seen among the workpeople. Nearly all cases developed the rash after several months on the job and although the dermatitis is eczematoid, a striking feature of an early case is that the rash is seen to originate from the follicles. The cause of the rash is, undoubtedly, the sodium carbonate in the carbon pellets; even when the carbon is washed after use it is still strongly alkaline to litmus. Carbon containing barium carbonate was not considered so harmful.

The new employee should be warned to report any rash immediately, the risk should be explained in terms of washing soda and no men allowed to do carbon packing straight after furnace work, when they are sweating. Frequent swilling of the arms with water, without proper drying, was a causative factor. Barrier creams were of doubtful value and gloves proved clumsy to work in. The only therapeutic measure of use in an established carbon packer's dermatitis is immediate change of job and the usual treatment for eczema is given.

A. Thelwall Jones.

Maximum Allowable Concentrations of Industrial Atmospheric Contaminants. COOK, W. A. (1945). *Industr. Med.*, **14**, 936.

A table is given of the maximum allowable concentrations of some 130 materials which may contaminate the atmosphere of workshops. Figures listed include those laid down by a number of states, by the United States Public Health Service, and by the American Standards Association. Based on these figures, on animal experiments, and on industrial experience, there is a final column with suggested maximum allowable concentrations expressed in parts per million and milligrams per cubic metre. The basis and reliability of each of the values given in this final column is recorded and there is included brief abstracts from the literature.

R. S. F. Schilling.

An Apparatus for Rapid Sampling of Large Air Volumes for Industrial Air Analyses. SILVERMAN, L., and WILLIAMS, C. R. (1946). *J. industr. Hyg.*, **28**, 21.

Large-volume air samples are sometimes required, e.g., in an investigation of radium dust risks an 1800 cu. ft. sample was necessary for satisfactory quantitative determinations. The apparatus designed allowed efficient sampling rates as high as 65 cu. ft. per minute. The air was drawn through a bell-mouth type of inlet, 1·12 inches in diameter, fitted to a 1·25-inch flexible tube, and the dust or fume was retained on the filter paper in the apparatus, which also contained the blower. When an ashless paper was not required the filter paper used was Eaton and Dikeman No. 613–21, and for gravimetric work, Whatman No. 44. To provide a maximum filtration area, the unit consisted of a cone and a frustrum of a cone, with a total area of 1·55 sq. ft. The paper was prevented from collapsing or bursting by a wire screen. The article is illustrated by three photographs of the apparatus, with its manometer and flow regulating valve.

H. M. Vernon.

Occupational Exposure to Arsenic in the Manufacture of Arsphenamine and Related Compounds. WATROUS, R. M., and MCCAUGHEY, M. B. (1945). *Industr. Med.*, **14**, 639.

Poisoning by inorganic compounds of arsenic is dealt with fully in all standard treatises on toxicology. The toxicology of the organic compounds of arsenic has been described as complications of therapy, but little has been written of the effects of occupational exposure to organic arsenical compounds.

The authors estimated concentrations of arsenical dust calculated as As_2O_3 in the atmosphere of various parts of a plant making arsphenamine and related compounds. The mean value of all manufacturing operations was 0·006 mg. per cu. ft., the mean for packaging operations was 0·0023 mg. per cu. ft. Concentrations ranged from 0·0002 to 0·017 mg. per cu. ft.

Urinary excretions were determined for workers in the manufacturing and packaging sections and for applicants for work. The ranges were as follows:—manufacturing 0·006 to 0·500 mg. per 100 c.cs. of urine; packaging 0·003 to 0·037; applicants (controls) 0·006 to 0·028. At higher levels of exposure in the manufacturing department increased urinary excretion was present but only a fraction of the calculated inhaled arsenic could be demonstrated in the urine. Analysis of hair in four manufacturing workers gave values higher than those previously reported in the literature, but these workers showed neither symptoms of arsenic poisoning nor unusually high concentrations of arsenic in the urine. The arsenic in the hair was believed to be of external origin and to have no diagnostic value. Symptoms of extremely mild arsenic poisoning were found among certain employees working under conditions of heavy exposure in the manufacturing department.

Results of 323 complete blood counts on 35 arsenical workers were compared with 221 complete counts on a control group of workmen not exposed to arsenical compounds. No statistically significant toxic effect on the bone marrow of those exposed to arsenical compounds was demonstrated.

R. S. F. Schilling.

Toxicology of 1, 2-Dichloropropane (Propylene Dichloride). I. Studies on Effects of Daily Inhalations. HEPPEL, L. A., NEAL, P. A., HIGHMAN, B., and PORTERFIELD, V. T. (1946). *J. Industr. Hyg.*, **28**, 1.

Dichloropropane ($CH_2Cl—CHCl—CH_3$) is widely used as a solvent and as an insecticide. In the present study various species of animals were exposed for seven hours daily, five days a week, in a chamber $4 \times 4 \times 6$ ft. 400 litres of air per minute were drawn through the chamber, or a complete air change every seven minutes. A concentration of 2200 p.p.m. (or 10·4 mg. per litre) proved to be very toxic. Eleven of the sixteen guinea-pigs exposed were dead by the end of the fifth exposure,

and five out of twenty rats died after five exposures or less. Of four rabbits, two were dead after the second exposure. All but one of eleven mice died before the completion of one exposure. At a concentration of 1500 p.p.m. most of the animals showed little evidence of being affected while in the chamber, though the growth of rats and guinea-pigs was adversely affected. Mice were very susceptible, all of twenty-two mice dying when exposed for four hours. At a concentration of 1000 p.p.m. twenty-five out of forty-five rats died after exposure on six to fifty-nine occasions, and three out of twelve guinea-pigs (twenty-two to ninety-seven occasions). Of five dogs and four pups, four died after twenty-seven to ninety-six exposures.

Many tests were made of liver and kidney function, but no abnormalities were discovered except a lowering of the plasma protein level, and this was probably due to voluntary starvation. Haematological studies gave negative results. Autopsies showed that guinea-pigs and rats had moderate to marked fatty degeneration of the liver, and moderate fatty degeneration of the kidney. Mice dying after a two-hour exposure to 1500 p.p.m. showed marked fatty degeneration of the liver. The four dogs which died after numerous exposures to 1000 p.p.m., showed moderate to marked fatty degeneration of liver, kidney, heart and adrenal gland.

When the toxicity of dichloropropane was compared with that of four other chlorinated hydrocarbons, it was found to be greater than all but one of them. Dichloromethane was the least toxic, repeated exposures of rats to 5000 p.p.m., over a period of six months, being well tolerated. Trichlorethylene was more toxic, only two out of six rats surviving repeated exposures to 3000 p.p.m. Carbon tetrachloride killed six out of thirty rats by the time that eight exposures to 2400 p.p.m. had been made. The most lethal hydrocarbon of all was dichloroethane, twenty-two out of twenty-eight rats being dead after fifteen exposures to 1000 p.p.m. These comparisons of the various chlorinated hydrocarbons are not exact, as different strains of rats and different diets were used. *H. M. Vernon.*

Exposures to Oxides of Nitrogen Accompanying Shrinking Operations. ADLEY, F. E. (1946). *J. Industr. Hyg.*, **28**, 17.

Welding operations incidental to the construction and repair of steel ships often produce distortion of the metal plates, especially on decks and bulkheads. Subsequent corrective measures include shrinking, which involves the application of heat to distorted sections of steel plate by means of an oxyacetylene flame. Shrinking is often necessary in small confined spaces, and the oxides of nitrogen produced may cause poisoning; e.g., a man exposed in this way died ten days later from chemical pneumonia. The acetylene torches used in the shrinking equipment commonly vary from a consumption of 60 cu. ft. to one of 250 cu. ft. per hour, and the temperature of the flame is about 6000° F. Samples of air were collected in the breathing zone of the unventilated compartment (of 600 cu. ft. capacity) in which the man was poisoned, and analysed by the phenol disulphonic method. A shrinking torch of 125 cu. ft. capacity was used. The only ventilation was through a single door, and eight air samples collected over a period of twenty-three minutes showed a concentration of oxides of nitrogen averaging 196 p.p.m., with a maximum of 480 p.p.m. The concentration was not appreciably affected by the water curtain surrounding the acetylene flame, nor by cool metal surfaces. It was found that (in a 700 cu. ft. compartment) the concentration of oxides of nitrogen varied with the size of the torch, being only 38 p.p.m. when the acetylene consumption was 16 cu. ft. per hour, 210 p.p.m. when it was 60 cu. ft., and 350 p.p.m. when it was 175 cu. ft. In relatively unconfined areas, with fair ventilation, the average concentration of oxides of nitrogen was only 4 to 48 p.p.m. *H. M. Vernon.*

Microdetermination of Mercury in Biological Material. CHOLAK, J., and HUBBARD, D. M. (1945). *Industr. Eng. Chem.*, **18**, 149.

A procedure suitable for determining minute quantities of mercury in biological and related materials is described. Organic matter is destroyed by digesting the sample in a special apparatus, by boiling first with a mixture of sulphuric and nitric acids, followed by a second boiling treatment after addition of potassium permanganate. Mercury is extracted from the decolorized acid-permanganate digest (diluted to 1·8 N. in respect to sulphuric acid) by means of a chloroform solution of di-β-naphthylthiocarbazone. The chloroform extract is then treated with a sulphuric acid-sodium thiosulphate mixture to separate the mercury from copper. The final estimation of mercury is made by photometering di-β-naphthylthiocarbazone-chloroform extracts of the copper-free aqueous phase following treatment with potassium permanganate. At least 50 g. of blood must be used for the reliable estimation of concentrations as low as 1 mg. of mercury in 100 g. of blood. A table, which compares the mercury content of the blood and urine of exposed persons, shows that the mercury content of the blood is very low. Except in one case, the amounts were well below 10 mg. for 100 g. of blood. The highest, 0·029 mg. per 100 g., was found in a case where the urine level was 4·44 mg. of mercury per litre. The mercury-di-β-naphthylthiocarbazone complex is more stable than the analogous dithizone complex.

A. Thelwall Jones.

Experimental Chronic Fluorine Intoxication. Effects on Bones and Teeth. BAUER, W. H. (1945). *Amer. J. Orthodont. and Oral Surg.*, **31**, 700.

A bitch was fed on a diet containing 0·4 g. of sodium fluoride daily. Some of a group of puppies between the 28th and 98th day were suckled by their mother fed on this diet, while others were fed by their mother for four weeks and then on food containing 0·1–0·2 g. of sodium fluoride. Extensive changes were produced both in the bones and developing teeth dependent on the dose and length of time for which the fluoride was given. Small doses, starting at an early age and administered for a long time, produced a dense periosteal bone formation. In puppies suckled throughout by their mother, bone changes were observed before changes occurred in the tooth buds; the periosteal cells assumed osteoblastic function before the enamel forming cells were affected. Changes in the bones and teeth could not be prevented by antirachitic diet. Calcification of excessive periosteal and endosteal bone in spite of continued administration of fluorine suggests that the pathogenesis of the lesions in bone are different in rickets and fluorosis. Fluorine produced changes similar to rickets in the bones and dentine of puppies but its effect on the bones of older dogs differed from that of rickets. The author considers a full investigation into X-ray changes in the skeleton of children living in endemic areas and showing 'mottled enamel' should be carried out. *K. M. A. Perry.*

The Thymol-Barbitone Test in Clinical Practice. MCCORD, C. P., KLINE, L. L., and WILLIAMS, P. E. (1945). *Industr. Med.*, **14**, 835.

The thymol-barbitone test first described by Maclagan in the *British Journal of Experimental Pathology* in 1944, is a serum turbidity test which, when positive, is said to indicate disturbed liver metabolism. The authors applied the test to 712 hospital patients, of whom 49 (6·8 per cent.) gave a positive reaction, and to 52 chemical workers exposed at apparently safe concentrations to various chemicals, some of which were known to be toxic. Of the latter group there were four positive reactors, but none of 30 presumed healthy individuals had a positive test. Liver damage was indicated in the greater number of reactors in the hospital patient series, but the authors state that in their investigations the validity of the test was lower than appraised by Maclagan.

R. S. F. Schilling.